

DISSERTATION
ON
ANALYSIS OF 50 CASES OF GANGRENOUS BOWEL DISEASE

Dissertation submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

*In partial fulfilment of the regulations
for the award of the degree of*

M.S. -GENERAL SURGERY- BRANCH – I



THANJAVUR MEDICAL COLLEGE,
THANJAVUR - 613 004.

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI - 600 032.

APRIL - 2013

CERTIFICATE

This is to certify that dissertation entitled '**A STUDY OF 50 CASES OF GANGRENOUS BOWEL DISEASES**' is a bonafide record of work done by **Dr. T. UMA MAHESWARI**, in the Department of General Surgery, Thanjavur Medical College, Thanjavur, during her Post Graduate Course from 2010-13. This is submitted in partial fulfillment for the award of **M.S., DEGREE EXAMINATION – BRANCH I (GENERAL SURGERY)** to be held in March 2012 under **The Tamilnadu Dr. M.G.R. Medical University, Chennai**.

Dr. M. ELANGO VAN, M.S.,
V.BALAKRISHNAN, M.S.,
Unit Chief
Department of surgery,
Thanjavur Medical College,
College,
Thanjavur.

Dr.
Professor and Head,
Department of surgery,
Thanjavur Medical
Thanjavur.

DEAN,
Thanjavur Medical College,
Thanjavur.

DECLARATION

I declare that this dissertation entitled '**A STUDY OF 50 CASES OF GANGRENOUS BOWEL DISEASES**' is a record of work done by me in the Department of General Surgery, Thanjavur medical college, Thanjavur, during my Post Graduate Course form **2010-13** under the guidance and supervision of my unit chief **Prof. Dr. M. ELANGO VAN, M.S.,** and Professor and Head of the Department of **Prof. Dr. V. BALAKRISHNAN. M.S.,** It is submitted in partial fulfillment for the award of **M.S., DEGREE EXAMINATION – BRANCH I (GENERAL SURGERY)** to be held in **March 2013** under the **Tamilnadu Dr. M.G.R. Medical University, Chennai.** This record or work has not submitted previously by me for the award of any degree or diploma from any other university.

THANJAVUR

Dr. T.UMA MAHESWARI.



THANJAVUR MEDICAL COLLEGE

THANJAVUR, TAMIL NADU, INDIA – 613 004

(Affiliated to the T.N. Dr. MGR Medical University,
Chennai)



ETHICAL COMMITTEE

CERTIFICATE

Name of the candidate : **Dr. T. UMA MAHESWARI**
Course : M.S (General Surgery)
Period of Study : May 2010 to November 2012
College : THANJAVUR MEDICAL COLLEGE
Dissertation Topic : ANALYSIS OF 50 CASES OF GANGRENOUS
BOWEL DISEASE

The Ethical Committee, Thanjavur Medical College has decided to inform that your Dissertation Topic is accepted and you are permitted to proceed with the above study.

Thanjavur


Secretary

Date:

Ethical Committee

ACKNOWLEDGEMENT

I thank the Lord God Almighty for giving me an opportunity to do my post graduation under the guidance of my chief Prof. Dr. M. ELANGO VAN, M.S., Thanjavur Medical College, Thanjavur. My heartfelt thanks to my chief for his advice and guidance in designing and enabling me to do this study with creative suggestions and constructive criticisms.

I am deeply indebted to Prof. Dr. V. BALAKRISHNAN. M.S., Head of the Department of surgery for being a source of inspiration and guidance.

I owe thanks to Prof. Dr. Manivannan, M.S., and Prof. Dr. Maragathamani. M.S., for their valuable advice and guidance.

I am grateful to Assistant Professors Dr. Prema latha, M.S., and Dr. Vimal, M.S., for their constant guidance and encouragement throughout the period of this study.

I thank all Assistant Professors, Department of Surgery, Thanjavur Medical College for their valuable suggestions.

I am very much thankful to my senior and junior post graduate for their valuable help and co operation.

I thank THE DEAN, Thanjavur Medical College, Thanjavur, for all the facilities and encouragement provided to me for the completion of my thesis in this prestigious institution.

I express my sincere thanks to all the patients, who in spite of their physical and mental sufferings have co operated to my study.

Special thanks to my family and my friends for their encouragement and constant support.

CONTENTS	PAGE NUMBER
INTRODUCTION	1
AIMS AND OBJECTIVES	2
REVIEW OF LITERATURE	3
MATERIALS AND METHODS	68
RESULTS AND OBSERVATION	71
DISCUSSION	90
CONCLUSION	96
BIBLIOGRAPHY	98
ANNEXURES	
➤ Proforma	103
➤ Master chart	106
➤ abbreviations	111

INTRODUCTION

Bowel gangrene is one of the most challenging surgical emergency with high morbidity and mortality all over the world. The causes are diverse and clinical and radiological manifestations vary according to etiology. Early diagnosis, prompt treatment and good postoperative care are the three important basic key stones for a successful outcome in a patient with bowel gangrene. These include rapid correction of fluid and electrolyte imbalance, control of sepsis and urgent & prompt resection of the diseased bowel. All will be lost once the lethal toxins enter the systemic circulation. The technical and judgment errors during surgery are the two important preventable factors that can have high impact on morbidity and mortality of the patient and therefore it should be limited. These errors can be minimized by meticulous surgery, clear goal and alternative surgical strategy- i.e. safest surgery that gives best outcome for each individual patient. However certain uncontrollable factors like advanced age, poor functional status and associated co-morbid systemic illness can cause potential life threatening consequences in a patient with bowel gangrene undergoing surgery.

AIM OF THE STUDY

- To analyze various etiological factors causing Bowel Gangrene.
- To evaluate the final outcome of the treatment offered.
- To study various factors influencing the morbidity and mortality associated with Bowel Gangrene.

REVIEW OF LITERATURE

DEFINITION

It is defined as hemorrhagic transmural necrosis of part or whole of bowel due to vascular compromise from variety of diseases.

CLASSIFICATION AND ETIOLOGY

According to the site of involvement it is broadly classified in to

1) Small bowel Gangrene

2) Large bowel Gangrene

The primary causes for impairment or insufficient blood flow to the intestines are many and by the nature of insult they are broadly classified in to three groups.

- Acute mesenteric vascular diseases
- Occlusive vascular diseases
- Non occlusive vascular diseases
- Acute intestinal strangulation
- Strangulated bowel resulting from hernia and adhesions

- Interrupted blood flow due to volvulus and intussusception
- Increased intra luminal pressure due to closed loop obstruction
- Others
- Trauma – physical,chemical,chemotherapy,radiotherapy.
- Neoplasm

Neoplasm can produce bowel ischemia in three ways

- By closed loop obstruction.
- Direct mechanical compressions of vessels
- Bacterial proliferation due to stasis of bowel contents proximal to the malignant stricture

ACUTE MESENTERIC VASCULAR DISEASES

Normally the bowel has extensive collateral circulation between celiac, superior mesenteric, inferior mesenteric and iliac arterial beds and there by protected from ischemic assault. Viability of bowel is endangered only when there is abrupt interruption of the blood supply to the gut .

MVD are classified in many ways as follows.

- 1) Acute or chronic in nature
- 2) Arterial or venous in origin
- 3) Transient or permanent interruption
- 4) Occlusive or non-occlusive according to etiology
- 5) Colonic or mesenteric according to site.

Incidence of acute colonic ischemia is about 50% to 60% and it is almost always due to arterial insufficiency. Whereas acute mesenteric ischemia contributes about 30 to 35 % and results from both arterial and venous insufficiency.

CAUSES OF MVD

CAUSES OF MVD

OCCLUSIONS

Thrombosis
Embolism

HEMODYNAMIC ALTERATION

Cardiogenic shock
Hypovolemic shock
Arrhythmias

TRAUMA

Blunt
Penetrating

IATROGENIC

Aneurysmal repair
Aorto iliac reconstruction

Pelvic surgeries

MEDICATIONS

Estrogen, Danazol
Psychotropic
agents
Cocaine,
Gold
Vasopressors

VASULITIS

Poly arteritis nodosa

SLE

Rheumatoid vasculitis

TAO

Takayasu's vasculitis

HYPER COAGULABLE STATE

Sickle cell anaemia
Protein C and S deficiency
Anti thrombin III deficiency

Polycythemia

OTHERS

Allergy
Long running
Portal hypertension

ACUTE INTESTINAL STRANGULATION

In any case of intestinal obstruction, when there is significant reduction in blood flow to intestines it will end in bowel gangrene. The possible mechanisms include

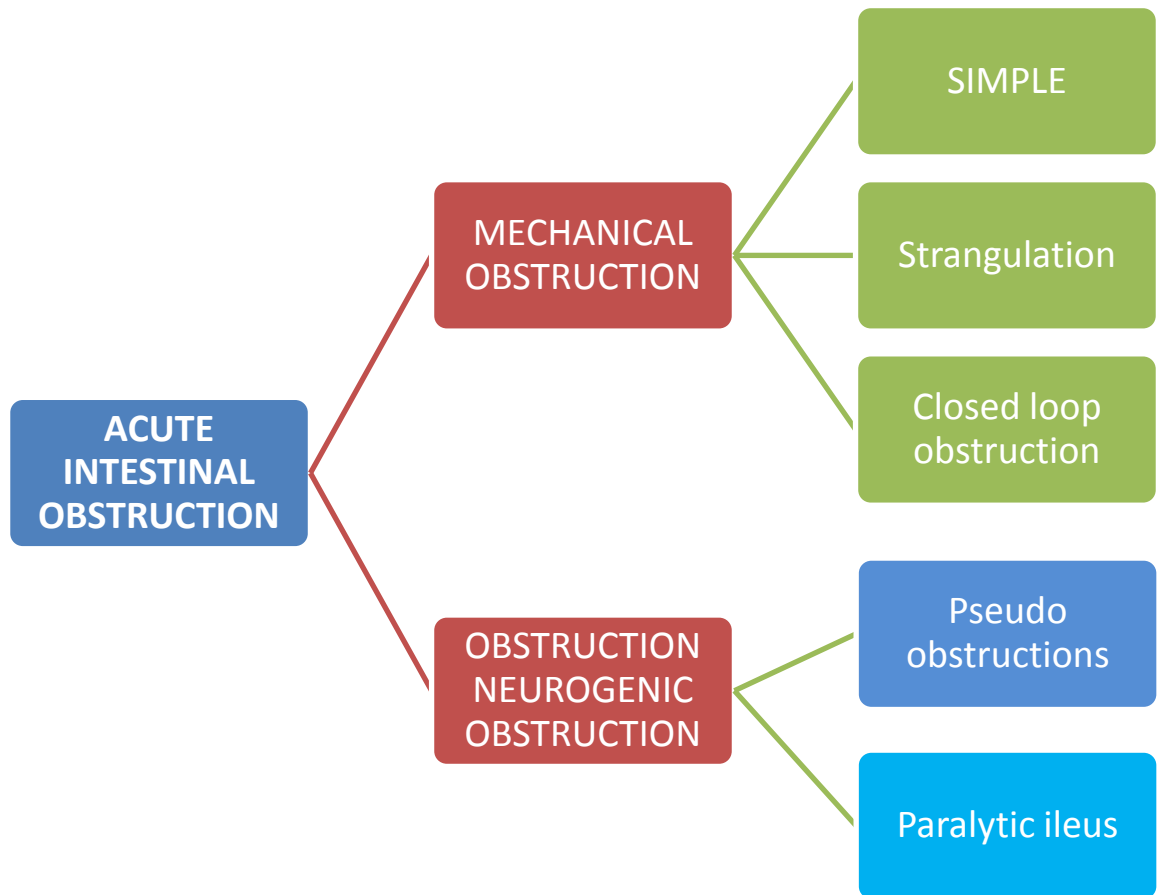
- Direct compression of mesenteric vessels by hernial orifice or axial twisting
- vascular compromise following increased intraluminal pressure from closed loop obstruction
- Associated local changes in intestinal wall.

Again acute intestinal obstruction is classified in many ways

- Dynamic or Adynamic obstruction
- Small or large bowel obstruction
- Acute or chronic
- Partial or complete
- Simple or strangulated or closed loop.

Acute, complete obstruction due to mechanical or functional intestinal obstruction with strangulation leads to bowel gangrene.

ACUTE INTESTINAL OBSTRUCTION



CLOSED LOOP OBSTRUCTION

Here both proximal and distal decompression is impossible due to obstruction at both ends. With a consequence there is rapid rise in intra luminal pressure secondary to accumulation of fluid and gas in the obstructed segment which causes marked hemorrhage and vascular compromise and if not treated early it will lead to bowel gangrene soon.

CAUSES

- Torsion of intestine around adhesive band.
 - Incarcerated bowel in hernia.
 - Volvulus of small /large intestine
 - Obstructive growth in colon with competent ileocecal valve

CAUSES OF MECHANICAL OBSTRUCTION

INTRA LUMINAL		INTRA MURAL		EXTRA MURAL	
Foreign body	Intra genic	Congenital	Atresia,	Adhesion /Bands	Post-operative
	ingested		stenosis		post inflammatory
	gall stone		duplication	Hernia	Internal
Intussusception	worms		diverticuli		External
			Diverticulitis		Annular Pancreas
Polyploid tumors				NASID	Malrotation
Impacted Fluids			Drug induced		Congenital
	Barium			KCL tablet	Omphalomesenteric duct remnant
		Inflammatory	Inflammatory diseases	Crohns	Anomalous origin of vessel
	Feces				Organomegaly
	Meconium		Infections	TB	Fluid collection
				Actinomycosis	
			Ischemia		Miscellaneous
			Radiation Benign		
		Neo Plasia	Carcinoma		
			Carcinoids		
		Trauma	Lymphoma		
		Miscellaneous	Sarcoma		
			Endometriosis		

CAUSES FOR ADYNAMIC OBSTRUCTION

NEUROGENIC	METABOLIC	PHARMACOLOGICAL
<ul style="list-style-type: none"> • Spinal cord Injury • Ureteric colic • Retro peritoneal tumor 	<ul style="list-style-type: none"> • Hypokalemic • Uraemia • $\text{Ca}^{++}, \text{Mg}^{2+}$ Imbalance • Hypothyroidism • Hypoparathyroidism • Diabetic ketoacidosis 	<ul style="list-style-type: none"> ➤ Anti-cholinergic ➤ Opiates ➤ Ganglion Blockers ➤ Tricyclic Anti depression ➤ Vincristine ➤ Lead poisoning ➤ Antacids
INFECTIONS		
<ul style="list-style-type: none"> • Systemic sepsis • Pneumonia • Peritonitis • Tetanus • Bacterial overgrowth • Herpes zoster 	MISCELLANEOUS	
	<ul style="list-style-type: none"> • Post-operative • Myocardial infarction • Renal Transplantation • Chemotherapy • Radiation 	

Post-operative ileus

Post-operative ileus may last for 0-24 hours in small bowels, 24-48 hrs.in the stomach, 48-72 hrs.in large bowels. It is due to increased sympathetic response to pain, Electrolyte imbalance, anaesthetic drugs and lack of motility and etc.

FUNCTIONAL OR ADYNAMIC OBSTRUCTION

It is mostly the consequence of enhancement of normal inhibitory mechanism of gut mobility due to local causes like infections and inflammations or systemic causes like sepsis, drugs.

In general Para sympathetic fibers are excitatory and sympathetic fibers are inhibitory to gut. Two types of intestinal inhibitory reflexes have been involved one is low threshold spinal reflex and the second one is high threshold reflex from paravertebral ganglia.

Small intestine has low level extrinsic and high level intrinsic cholinergic control and sympathetic inhibitions is mediated by alpha adrenergic fibers. Whereas colon has less cholinergic drive than small bowel and sympathetic inhibition is mediated by beta-adrenergic fibers.

Ileus is a neurological phenomenon resulting from both spinal and peripheral reflexes, secondary to reflex stimulation of adrenergic neurons. In contrast colonic obstruction is due to abnormality in either muscular or intrinsic neural system.

Neurogenic component is due to diminished parasympathetic stimulation and inhibition of sacral parasympathetic path way.

PATHO PHYSIOLOGY OF ILEUS AND PSEUDO-OBSTRUCTION

Ileus	Pseudo obstruction
Acute process	chronic process
Neurogenic Phenomenen	Both Neurogenic +MascuarPhenomenen
Sympathetic stimulation	Para sympathetic inhibitions

EPIDEMIOLOGY

In worldwide, depending upon ethnicity, age, dietary habits, and geographical locations, there are wide variations in frequency and etiology of bowel gangrene.

Overall incidence of small bowel gangrene is about 70% and that of large bowel is about 30%. Small bowel gangrene most often results from hernia, adhesions and mesenteric insufficiency. Large bowel gangrene is from malignancy, volvulus, and diverticulum. Now a day the incidence of obstructed hernia is decreased due to elective hernia repair and increasing incidence of pelvic and abdominal surgeries increases the incidence of adhesive obstruction.

Cause	Small bowel gangrene	Large bowel gangrene
MVD	30-35%	50-60%
Intestinal strangulation	85%	15%

Both MVD and intestinal strangulations are common among geriatric population and the mean age group is around 50 yrs of age. Bowel gangrene in younger population most often results from vasculitis and adhesive

intestinal strangulation whereas in geriatric population most often results from thrombo embolism, hernia, malignant obstruction and volvulus.

AGE DISTRIBUTION

AGE	MVD	INTESTINAL STRANGULATIONS
Above 50yrs	Thrombo embolism	Hernia malignancy, diverticuli, volvulus
Below 50yrs	Vasculitis Drugs Coagulopathy	Adhesions bands

Overall both males and females are more or less equally affected by bowel gangrene. Among the individual cases, all types of hernias are common among male patients and adhesive obstruction is common among female patients due to increased incidence of PID and Gynecological surgeries.

PATHOPHYSIOLOGY OF BOWEL GANGRENE

Pathophysiology of MVD is different from that of intestinal strangulation and hence both are discussed separately.

PATHOPHYSIOLOGY OF MVD

Splanchnic circulations receive approximately 25% of resting cardiac output and this increases by about 10% during post prandial period. Mucosal and sub mucosal layer receives about 75% of the blood flow. Splanchnic circulation is regulated by intrinsic and extrinsic regulated system cellular and molecular level. Intrinsic system is mainly mediated by metabolic and myogenic pathway. The extrinsic regulatory system is mediated by neural and hormonal factors.

INTRINSIC REGULATORY SYSTEM

- **Metabolic pathway**

When there is imbalance between oxygen supply and demand accumulation of local metabolites like H^+ , K^+ , CO_2 , occurs which causes vasodilatation

- **Myogenic pathway**

Acute decrease in the splanchnic blood flow causes relaxation of the vascular smooth muscle.

EXTRINSIC REGULATORY SYSTEM

- **NEURAL PATHWAY**

Sympathetic adrenergic system especially alpha adrenergic system and cholinergic system are responsible for resting tone of splanchnic arteriolar system and when there is reduction in blood flow there will be increased vasoconstriction in both pre capillary arteriolar bed and post capillary Venous bed. Thereby blood flow to the gut is preserved at or near normal levels

In addition adrenergic system changes bowel mobility, wall tension and absorption, all of which can have a pronounced effect in the regional blood flow.

- **HORMONAL MECHANISM**

Vasopressin and angiotensinII are the 2 important hormones which are responsible for regulations of the blood flow in response to fall in the blood pressure.

- **PATHOLOGY OF MVD**

The basic pathological processes behind bowel gangrene in MVD are as follows

- Persistent and prolonged mesenteric arterial vasoconstriction
- Cellular injury

- Tissue hypoxia
- Reperfusion injury.
- Disruption of mucosal barrier and bacterial translocation

Persistent vasoconstriction

Initially vasodilatation and auto regulation maintains the blood flow for a brief period. When the ischemia is prolonged vasoconstriction develops in the involved vascular bed and elevating the blood pressure in the distal bed and ultimately causes reduction in collateral flow and potentially compromises the bowel viability

CELLULAR INJURY

When the intestine is deprived of an adequate blood supply for more than 30 minutes extensive changes occurs in the cellular level including accumulation of fluid between the cells and the basement membrane. Tips of the villi begin to slough followed by necrosis of cellular membrane. This results in inflammatory reactions followed by bacterial proliferation, ultimately leads to cellular death which progresses from the lumen outwards until there is transmural necrosis of bowel wall.

REPERFUSION INJURY

It occurs when the flow is reestablished after an attack of ischemic insult. It is mainly mediated by endogenous substances such as oxygen free radicals, platelet activating factor, arachidonic acid metabolites and bacterial endotoxins.

MUCOSAL BARRIER DAMAGE AND BACTERIAL TRANSLOCATION

Both ischemic and reperfusion injury causes mucosal damage and altered the mucosal permeability resulting in translocation of luminal bacteria in to mesenteric lymph node and systemic circulation.

All together ends in trans mural necrosis and bowel gangrene

PATHO PHYSIOLOGY OF INTESTINAL STRANGULATION

The sequence of events in strangulated bowel is as follows

- **Compression of veins**

Accumulation of gas and fluid proximal to the obstruction causes luminal distension. Resulting intra luminal pressure causes venous compressions and edema. At this stage bowel and mesentery looks congested and blue in color.

- **Compression of arteries**

Increased capillary pressure, capillary leakage leads to accumulation of fluids and RBC's in the wall and the lumen. This ultimately causes arterial compression. At this time the bowel is oedematous, losses its glistening appearance, mucous membrane become ulcerated. And gangrene is imminent

- **Retrograde thrombosis of mesenteric vessels**

Stasis of blood flow along with increased capillary permeability causes thrombosis in mesenteric vascular system – both arterial and venous system which further proceeds in retrograde manner

- Bacterial over growth and translocation of bacteria with ina few hours of strangulation bacterial overgrowth and release of vasoactive release of toxins occurs in the involved segment resulting in mucosal ischemia and necrosis. It is followed by translocation of the bacteria in to this systemic circulation this ultimately leads to gangrene, perforation sepsis

CLINICAL FEATURES OF BOWEL GANGRENE.

Clinical features of bowel gangrene may vary according to various causes, severity and complications like shock, sepsis.

Various mode of presentation of bowel gangrene include

- 1) Features of acute intestinal obstruction
- 2) Features of hypovolemic shock
- 3) Features of peritonitis
- 4) Features of sepsis and its complications

FEATURES OF ACUTE INTESTINAL OBSTRUCTION:

Abdominal pain, vomiting, distension and obstipation are the four cardinal symptoms of Bowel Gangrene.

ABDOMINAL PAIN:-

Crampy intermittent abdominal pain is the initial complaint which corresponds with hyper motility. Initially the pain is diffuse and poorly localised. But generally it is felt at the centre of abdomen in small bowel involvement and lower abdomen in large bowel involvement. And also in small bowel involvement this paroxysmal pain occurs in short interval. Where as in large

bowel involvement , this pain is spaced farther apart in time and duration. In a case of Functional obstruction pain is mild, diffuse and there is no such waves of colicky pain. In case of MVD, the nature of the pain is often acute severe pain out of the proportion to physical finding and often patient had H/O post prandial pain in the week to months prior to the present history.

In the late stage, once the bowel becomes gangrene, initial intermittent colicky pain becomes a **CONSTANT PAIN**.

VOMITING

The severity of vomiting and the frequency of vomiting is more in high obstruction and it is less in a case of lower obstruction. Higher obstruction is characterized by bilious vomiting and lower obstruction is characterized by feculent vomiting.

OBSTIPATION

Luminal contents distal to the lesion may be passed initially. Once the distal segment becomes empty complete obstipation(that is failure to pass gas and stool) is the essential feature in Bowel Gangrene. Exceptions are mesenteric ischemia, acute intussusception where there may be Red currant jelly stools / hematochezia.

ABDOMINAL DISTENSION

Accumulation of gas and fluid proximal to the obstruction causes distension of involved segment. The severity of distension depends upon the level of obstruction. It is minimal in high obstruction and more prominent in low obstruction.

CLINICAL SIGNS Of acute intestinal obstruction are :

- Abdominal distension
- Diffuse tenderness
- Absent Bowel sound
- Signs of dehydration –
- Dry skin and dry tongue, Sunken eyes

BOWEL SOUNDS :

High pitched tinkling “Bowel sound” is characteristic for paralytic ileus and Prolonged rapid, high pitched Bowel sounds with burst, is characteristic for early mechanical obstruction. Absent Bowel sounds indicates Bowel Gangrene.

CLINICAL FEATURE OF HYPOVOLEMIC SHOCK : -

Patient become anxious, pale. Tachycardia, hypo tension and oliguria are the three cardinal features of hypovolemic shock.

CLINICAL FEATURE OF PERITONITIS :-

Once peritonitis sets in :

- Abdomen become rigid, not moves with respiration.
- Rebound tenderness and Guarding
- Bowel sounds absent.

CLINICAL FEATURES OF SEPSIS : -

Rise in Temperature indicate Localised Sepsis and Hypothermia and Hypotension indicates systemic sepsis.

But none of these findings are present in 10 – 15% of patient with Bowel Gangrene and atleast one of the finding is present in 90% of cases with simple obstruction. Thus , Diagnosis of bowel gangrene on the basis of clinical findings alone is difficult.

DIAGNOSIS

It is very important to elicit detailed history to arrive the diagnosis & to establish the cause. Confirmation is by various forms of imaging procedure.

H/o constant abdominal pain, rebound tenderness and involuntary guarding, absent bowel sounds, Fever, tachycardia, and Hypotension, leucocytosis are the cardinal features of Strangulation.

H/o Previous surgery points towards the etiology of adhesive obstruction.

H/o Recurrent attacks of sub acute intestinal obstruction without any previous

H/o surgery suggest the possibility of chronic intra abdominal inflammatory conditions / Neoplasia.

H/o Elderly age group. H/o co-morbid illness like atherosclerotic diseases, H/o

Postprandial pain, and acute severe abdominal pain with less signs, H/o taking certain drugs which precipitates mesenteric ischemia all in favor of mesenteric occlusive diseases.

H/o long standing hernia with recent onset of irreducibility suggests bowel strangulation at hernia site.

LABORATORY TESTS

FEATURES OF FLUID AND ELECTROLYTE LOSS : -

- Hb – Elevated due to haemoconcentration.
- PCV- Elevated due to haemoconcentration
- Urine – Specific Gravity 1.025 to 1.030 mild proteinuria
- Blood Urea – Increased due to prerenal failure.
- Serum Creatinine- Increased due to prerenal failure.
- Serum Electrolytes- Na^+ , K^+ , Cl^- , HCO_3^- decreased.

FEATURES SPECIFIC TO BOWEL ISCHEMIA: -

- Leucocytosis
- Increases in serum amylase, inorganic Phosphate, Hexosaminidase,
- intestinal fatty acid Binding protein
- Serum D lactate levels.

These tests may be helpful in diagnosis of established bowel infarct but none in a case of imminent bowel gangrene.

RADIOGRAPH AND IMAGING

Plain radiograph of abdomen in Erect and supine position is sufficient to confirm the diagnosis of Bowel Gangrene. More sophisticated imaging procedures like CT / MRI may lead to unnecessary delay in resuscitation and treatment and is needed only in very few patients with uncertain diagnosis and etiology.

INTERPRETATION OF PLAIN X RAY

Air under diaphragm in erect x ray indicates Bowel Perforation.

Multiple air fluid levels in Erect x ray, dilated loops in supine x ray , with absence of gas distal to obstruction denotes obstruction .

a.Small Bowel is considered as dilatation when the diameter is more than 3 cm.

b. Proximal colon - more than 8 -10 cm.

c.Sigmoid colon – more than 4 to 5 cm.

Characteristic features of Gas shadows of small and large Bowel obstruction.

X-RAY FINDINGS IN SMALL BOWEL AND LARGE BOWEL

OBSTRUCTION

SMALL BOWEL	LARGE BOWEL
Tends to lie in centre of abdomen, more or less in transverse lie, with step ladder pattern.	Tends to lie at the periphery.
Jejunum characterized by Regularly spaced Valvulae conniventes that traverses full width of bowel.	Haustral folds are irregularly spaced and traverse only part of the width of Bowel.
Indentations present. Ileum is characterless. Cecum is identified by rounded shadow in right lower abdomen.	Indentations are not present.

FEATURES OF CLOSED LOOP OBSTRUCTION: -

Usually Closed loop contains very little gas and is completely filled with fluids; hence, it is very difficult to diagnose closed loop obstruction by plain abdominal x ray.

FEATURES OF PARALYTIC ILEUS:-

- Air fluid level is prominent throughout the Bowel.
- Gas shadows present diffusely both in small bowel and colon.
- Diaphragm is elevated.

FEATURES OF BOWEL GANGRENE:-

- Thickening of Bowel wall.
- Loss of mucosal pattern and thumb printing appearance.
- Loop fixation with loss of valvulae connivantes.
- Air in the bowel wall, mesenteric veins and portal vein.
- Free air under diaphragm in case of perforation.

OTHER IMAGING PROCEDURES

Contrast studies are contra indicated in suspicious cases of Bowel Gangrene.

Computed Tomography and Ultra sound abdomen are useful in patients with suspected malignancy, no previous surgeries, and signs suggesting of intra abdominal inflammation. Otherwise routine CT abdomen is not needed in all cases.

CT FINDING IN BOWEL GANGRENE

- Bowel wall thickening
- Luminal stenosis
- Intra mural and extra intestinal air

- Fluid filled intestine.
- Other fluid collection
- Mesenteric arterial and venous thrombo- embolism.

MANAGEMENT

GENERAL CONSIDERATIONS IN MANAGEMENT OF THE PATIENT WITH BOWEL GANGRENE

Diagnosis and Management should be done as quickly as possible. Otherwise it may lead to high morbidity and mortality.

Hence the overlapping sequences of events in managing the patients with Bowel Gangrene should be investigation, resuscitation and surgery.

Four Measures that are combating and overcoming the effect of bowel gangrene are: -

- Rapid and adequate correction of fluid and electrolyte imbalance.
- Decompression of distended bowel.
- Control of sepsis with liberal use of broad spectrum antibiotics.
- Surgical dealing of underlying Pathology. Resection of Gangrenous part of Bowel and restoration of Bowel continuity.

FLUIDS AND ELECTROLYTE REPLACEMENT

The Goal of the treatment includes

- Restoration of Volume deficit

- Correction of electrolyte abnormality.
- Correction of acid base imbalance.
- Correction of other metabolic derangement.

This is done by rapid infusion of large amount of isotonic crystalloid solutions like Normal saline or Ringer lactate Up to 3.5 liters of Isotonic saline is needed if there is severe dehydration.

Patients with severe and prolonged vomiting commonly have hypokalemic metabolic Alkalosis. Hence, Potassium and Chlorides should be measured and replaced appropriately. But before giving potassium infusion, the urine output and Renal function should be adequate.

Rate of fluid administration can be monitored by

- Hourly Urine output, if the patient is hemodynamically stable.
- Central venous pressure if the patient is hemodynamically unstable or in the presence of renal, cardiac or pulmonary diseases complications.

(2)DECOMPRESSION OF DISTENDED BOWEL

It is very important to decompress the bowel

- To prevent further distension of bowel with swallowed air
- To prevent aspiration while vomiting or on induction of anesthesia.

- To improve the ventilation by relieving the abdominal distension.
- To remove the highly toxic intra luminal contents before surgery.

So, whenever the patients come with abdominal distension, the patient should be put on Nil per Oral and Nasogastric decompression by 16-18 French rylestube.

CONTROL OF SEPSIS

Following intestinal strangulation, there is rapid bacterial overgrowth of both aerobic and anaerobic type of flora and translocation of Bacteria in to the systemic circulation. To combat this, liberal use of broad spectrum antibiotic is mandatory. It is usually covered by II/ III generation cephalosporin or combination of aminoglycoside and metronidazole if renal function is good.

SURGICAL MANAGEMENT

The surgical dictum holds that avoidable preoperative complications arise as a result of technical or more judgment errors.

AIM OF SURGICAL MANAGEMENT OF BOWEL GANGRENE INCLUDES

- **DEALING OF THE UNDERLYING PATHOLOGY**
 - To restore the blood flow in a case of MVD, either by thromb embolectamy/ bypass graft.
 - To relieve obstruction in a case of strangulated bowel. Ex:- Adhesion release, hernia Repair.
- **RESECTION OF GANGRENOUS BOWEL**
- **RESTORE THE BOWEL CONTINUITY EITHER BY**
 - Primary anastamosis with or without proximal stoma if the conditions favor.
 - Diversion procedures and secondary anastamosis later Ex: - colostomy, ileostomy, Hartman's procedure

PRINCIPLES OF EXPLORATIVE LAPARATOMY IN BOWEL GANGRENE

- Incision should be adequate enough to expose all four quadrants of abdomen; it is usually offered by long vertical midline incision.
- Initial Assessment directed towards
 - Site of lesion
 - Nature of Lesion
 - Viability of Bowel

Identification of decompressed bowel below the diseased bowel indicates the point of pathology. In General, if cecum is collapsed, the pathology is in small bowel, and dilated cecum indicates large bowel pathology.

Differentiation between viable and non viable intestine during surgery

SL.NO.	FEATURES	VIABLE	NON VIABLE
1	Colour	Red and dark purple	Green / Black discoloration
2	Mesentery	Oedematous . but arterial pulsations preserved	Loss of pulsations in mesentery
	Thrombosis	No thrombosis	Thrombosis in mesenteric vessels
	Bleeding	Mesentery bleeds on pricking	No Bleeding or pricking.
3	Visceral peritoneum	Normal shine preserved	Loss of normal shine
4	Peristalsis	Peristalsis present	Absent peristalsis
5	Response to towels wrung in warm saline and administration of 100% oxygen.	Return of normal peristalsis appear	No Return of normal color

- Intra operative decompression of Bowel is essential for adequate exposure, Optimal bowel viability and proper abdominal closure. More over if obstruction is relieved without proper decompression, the toxic substances in affected segment may pass on to the normal bowel and rapid absorption of toxic substances can occur and may lead to septicemia manual decompression.

It can be done by three ways: -

- Manual retrograde decompression
- Passage of Long naso intestinal tube
- Controlled enterotomy.

Manual retrograde decompression is the safest ,quickest procedure, but care must be taken while handling the infarcted bowel.

DEALING OF THE UNDERLYING ETIOLOGY WHICH CAUSES BOWEL OBSTRUCTION

Ex: Release of adhesion, restoration of blood flow in case of MVD

Revascularization should precede the evaluation of intestinal viability. Because the bowel that initially appears non viable may recover after restoration of adequate blood flow.

(5) RESECTION OF IRREPARABLY DAMAGED BOWEL

Non viable bowel should be identified and resected. Frank Gangrene can be identified clinically as discussed earlier. In doubtful cases, bowel viability can be assessed

- Surface fluorescence
- Perfusion flurometry

- Doppler ultrasonograph

If it is not available or still it is confusing, it is better to resect the clearly necrotic bowel and bring the resected ends to the surface and construct the stoma. The other option is second look surgery within 12 hours after adequate supportive measure to improve viability of bowel.

(6)PRIMARY ANASTOMOSIS

Intestinal anastomosis are fashioned in many ways like hand sewn / stapler etc., but in emergency surgeries, hand sewn technique is the choice and hence discussed in details. Anastomosis can be done either by

- Two layer or single layer
- Running or interrupted manner
- Absorbable or non absorbable suture materials
- End to end or End to side or Side to side anastomosis

IN TWO LAYER ANASTOMOSIS

- Inner layer is anastomosed by full thickness running sutures with $3^{0}/4^{0}$ absorbable suture material.

- Outer layer is anastomosed by investing seromuscular suturing with non absorbable / absorbable suture, either by interrupted / continuous stitch.

IN SINGLE LAYER ANASTOMOSIS

Hence, single layer of full thickness stitch with absorbable / non absorbable suture, either by continuous interrupted manner. Whatever may be the technique, ultimate goal is

- To prevent leakage
- To promote healing
- To preserve Bowel length
- To prevent stricture formation.

TECHNICAL CONSIDERATION IN BOWEL ANASTOMOSIS

- Strict aseptic precaution is essential to prevent anastomotic leak. So, in the presence of sepsis, it is better to avoid primary anastomosis.
- Adequate mobilization, tension free approximation of well vascularised bowel, Inversion of mucosal edge into the lumen, incorporation of sub mucosa in the closure , all these four techniques are essential for good healing.

Inadequate perfusion and tension across the anastomosis will lead to anastomotic leak in early stage and stricture and obstruction in late stage.

CRITERIA FOR PERFECT ANASTOMOSIS

- ANASTOMOSIS should be water tight
- Must be tension free
- Should have adequate perfusion
- Must have mucosal approximation and sub mucosal incorporation.

ANASTOMOSIS or STOMA

Primary anastomosis is not feasible in following condition

- (1) If the patient is hemodynamically unstable during surgery.
- (2) The vascularity of the resected margin is doubtful.
- (3) Proposed anastomosis will be under tension
- (4) In the presence of fecal or purulent material in the peritoneal cavity.

In such condition, it is better to perform diversion procedure,. They are:

- (1) Colostomy or Enterostomy or according to site
- (2) Loop or End stoma according to technique

GENERAL PRINCIPLES IN STOMA CONSTRUCTIONS:

- (1) Temporary stoma is usually fashioned as loop stoma.
- (2) Permanent stoma is usually fashioned as End stoma.
- (3) Ileostomy is sprouted but a colostomy is flush.
- (4) Ileostomy usually constructed in right Iliac fossa.
- (5) End colostomy usually constructed in left iliac fossa.
- (6) Temporary transverse loop colostomy constructed in right upper
Quadrant.

SPECIAL TYPES OF BOWEL OBSTRUCTION

BOWEL OBSTRUCTION DUE TO ADHESIONS / BANDS

Definition: Abnormal connective tissue attachment between tissue Surfaces.

Causes: (1) congenital bands

(2) Acquired -- Post inflammatory

CAUSES FOR ADHESIVE OBSTRUCTION

POST OPERATIVE	POST INFLAMMATORY
Ischemic Areas : Site of Anastomosis Reperitonealisation of raw area	Infection Peritonitis TB
Foreign Body Talc, Starch, etc.,	Inflammation Crohn's disease Radiation Drugs - Propranolol

EPIDEMIOLOGY:

Currently post operative adhesion is the leading cause of bowel obstruction and strangulation in the western world, where there is increased no. of abdominal surgeries when compared to past. Though, any type of Laparotomy can produce adhesion, it is more common among the pelvic surgeries. In long term follow up about 5% of patients undergoing surgeries will develop adhesion obstruction. Among those 10- 30% will suffer from additional episodes.

PATHOPHYSIOLOGY

Increased outpouring of fibrin resulting from peritoneal injury and decreased activation of tissue plasminogen activator (tPA) resulting from serosal injury are the primary pathological factors responsible for adhesion formation.

There are four types of adhesions:

Type I adhesion:

- Post operative **FIBRINOUS** adhesion
- It occurs usually between 3rd to 6th Post operative day
- Made up of fibrin, only flimsy adhesion.
- Common after pelvic surgeries.
- Usually disappears after some time.

(2)TYPE II ADHESION

(POST OPERATIVE FIBROUS ADHESION).

- It produced strong Band
- Common cause of post operative obstruction

(3)TYPE III

Adhesion of loop of intestine to an inflamed intra peritoneal structure.

(4)TYPE IV

Adhesion followed by chemotherapy / Radiotherapy

PREVENTION OF ADHESION

- Meticulous surgical handling and technique
- Perfect hemostasis
- Thorough toileting of peritoneal cavity
- Careful inspection and removal of foreign body in peritoneal cavity
- Covering the anastomosis and raw peritoneal surface
- Use of monofilament suture for fascial closure
- Avoidance of closure of peritoneal closure as a separate layer

DRUG PROPHYLAXIS

Drugs like anticoagulants, dextrans, anti histamine, NSAIDS, povidone, streptokinase and hyaluronate barriers can be used to prevent adhesions. But its efficacy is not yet proved and not recommended in routine practice.

CLINICAL FEATURES

Previous History of surgery, disease like TB, Crohn's disease, PID and history of recurrent attacks of subacute intestinal obstruction and presence of laparotomy scar, altogether with radiological evidence points towards adhesive pathology. Otherwise clinical features and radiological features are those of classical acute intestinal obstruction with bowel gangrene.

TREATMENT

Lysis of Adhesion and resection of gangrenous bowel and anastomosis.

PRINCIPLES OF ADHESIVE LYSIS

- Abdomen may be opened through previous undisturbed abdominal wall.
- Transverse incision is safe in patients who have multiple vertical laparotomy scars.

- Limiting the division only to the causative adhesion, leaving behind the quiescent adhesion undisturbed.
- Because, division of Quiescent adhesion will only lead to further adhesion.
- Cover the bare area with omental graft.

MANAGEMENT OF RECURRENT ADHESIVE OBSTRUCTION

- Noble plication in which bowel loops are sutured
- Child Philips, trans mesenteric plication
- Intra luminal stenting with long intestinal tube

BOWEL GANGRENE DUE TO STRANGULATED HERNIA

Definition:

It is defined as protrusion of part or whole of viscous through an abnormal opening in the wall of containing cavity.

EPIDEMIOLOGY

Strangulation of bowel in various type of hernia is the second most common cause of bowel gangrene in the western world. Approximately 5% of external hernia will undergo emergency surgery.

CLASSIFICATION		
EXTERNAL HERNIA	INTERNAL HERNIA	POST OPERATIVE HERNIA
Inguinal	Foramen of Winslow	Incisional hernia
Umbilical	Defect in broad ligament	Stoma related
Epigastric	Diaphragmatic hernia	Post surgical mesenteric defect
femoral	Paraduodenal hernia	
Obturator	Retrocecal hernia	
sciatic	Inferior sigmoid fossa	
Lumbar		
spigelian		

PRECIPITATING FACTORS

- Presence of pre formed sac
- Weakening of musculature – obesity , old age
- Increased intra abdominal pressure
- Chronic cough
- Chronic constipation
- Lower urinary tract obstructive symptoms
- Pregnancy
- Intra abdominal mass, ascites

CLINICAL FEATURES

All types of hernia are more common in Males than females

- Usually presented with long history of hernia with recent onset of irreducibility followed by acute signs and symptoms of bowel obstruction and strangulation
- Tense, tender irreducible hernia with absence of cough impulse and bowel sound strongly suggests strangulation.

TREATMENT

Resection of strangulated bowel and anastomosis followed by hernia repair.

PRINCIPLES IN MANAGEMENT OF STRANGULATED HERNIA

Exposure and control of hernia sac, its contents and constricting site

- Release of constricting agent by division
- Sac opened and contents released
- Resection of gangrenous bowel and anastomosis
- Repair of hernia after transfixing the neck of sac

BOWEL GANGRENE IN STRICTURE

CAUSES OF STRICTURE	
BENIGN	MALIGNANT
Post anastomotic	lymphoma
TB	Sarcoma
Crohn's disease	Carcinoma
actinomycosis	
Drug induced	

Clinical presentation is as like that of any intestinal obstruction with strangulation.

MANAGEMENT

- Resection of Gangrenous bowel along with stricture part and anastomosis.
- In any case **never do by pass** and anastomosis as it may lead to blind loop syndrome.

BOWEL GANGRENE DUE TO INTUSSUSCEPTION

Definition : -

it is defined as invagination of proximal gut into the distal gut resulting in obstruction followed by ischemic injury to bowel.

EPIDEMIOLOGY

Infants under two years of age are more prone for intussusception.

Intussusception in adults is only about 5%

PATHOPHYSIOLOGY

Pathology in adult is different from that of children and 90% of adult patients are associated with some pathological leading point either benign or malignant tumor.

Benign lesion such as submucosal lipoma, diverticulum, polyps, leiomyoma are the main leading points in small bowel intussusception. Whereas malignant lesion are common in large bowel.

Post operative intussusception are related to sutureline (20%), adhesion 30% to intestinal tubes.

TYPES OF INTUSSUSCEPTION

- Entero – enteric
- Ileo – colic
- Ileo – ileal
- Colo – colic

in adults colo – colic intussusception is more common.

CLINICAL FEATURES

Sudden onset of acute abdominal pain which occurs intermittently with appearance of lump during the peak of an attack and passage of red currant jelly stools is the classical picture of intussusception.

RADIOLOGICAL FEATURES

No specific features could be made out in plain x ray except multiple fluid levels. Barium enema shows claus sign but contra indicated in the presence of bowel gangrene. CT scan extremely useful to detect the involved segment and a mass lesion from intussusception. Three concentric circles that form as one segment of bowel invaginates into another and central circle by entering layer of intussusception and 2nd circle by entrapped mesentery and 3rd represents the intussusciens .

TREATMENT - Enbloc Resection And Anastomosis Is The Treatment Of Choice

ILEO SIGMOID KNOTTING

A loop of ileum wraps around the base of an elongated sigmoid colon or vice versa, and can strangulate either one or both bowel segments. It is a variant of midgut volvulus. It is otherwise called as compound volvulus.

ETIOLOGY

May be due to genetic, dietary, habitual. In south india, labour class people drink large quantities of GANJI, a preparation containing jowar, buttermilk and water may predispose. An abnormally long sigmoid colon with a lean and lengthy ileal mesentery.

CLINICAL FEATURES

Abrupt onset of obstructive symptoms with rapid deterioration of general condition is the clinical picture. Incidence of ileal gangrene is about 80% and sigmoid is about 20%.

INVESTIGATIONS

- Shows distension of both ileal loops and sigmoid colon

- Disproportionately dilated loops of the bowel with their limbs directed downwards into their lower quadrants.
- Small bowel air fluid level.
- Undistended but fecal loaded bowel proximal to the pelvic colon
- Medial deviation of the descending colon.

TREATMENT

Emergency laparotomy, untwisting of ileum and resection of gangrenous bowel and anastomosis.

BOWEL GANGRENE CAUSED BY SIGMOID VOLVULUS

Definition :

Volvulus is defined as axial twist or rotation of portion of alimentary tract about its mesentery in such a way as to obstruct the lumina of both proximal and distal loop of the segment(closed loop obstruction) and lead to vascular compromise and early bowel gangrene.

TYPES

- Primary volvulus

It is due to congenital mal rotation of gut. Abnormal attachment of mesentery and congenital band. Ex:- sigmoid volvulus , cecal volvulus

- Secondary volvulus

It occurs along the acquired adhesions or stoma.

Most common site of volvulus is , sigmoid 75% , cecal volvulus -25%

PREDISPOSING CAUSES

- Band of adhesions (peridiverticulitis) either tethering the base of the two limbs closed together or fixing the apex.
- Overloaded pelvic colon
- Long freely movable sigmoid loop on a long and freely movable mesosigmoid
- Acquired mesocolon
- Narrow attachment of pelvic mesocolon
- The absence of the last band of lane and creation of wide left paracolic gutter
- Fibrosis in the middle of mesosigmoid (intermediate group of lymph node) narrowing the base and creating a short of pedicle.

- The lateral limb of the mesosigmoid is always in approximation with ovaries in females but it has a very variable disposition in male extending from internal inguinal ring to the left iliac crest, Hence this horizontal upper limb, which is seen in males, predisposes to volvulus.

PATHOLOGY

In most cases of sigmoid volvulus the upper limb of the loop descends in front of the lower, twisting on its mesenteric axis from one and a half to two turns in anticlockwise direction. The loop may rotate half a turn in which event spontaneous rectification can occur. After one and a half turn rotation the veins involved in the torsion are compressed. If it rotates more than one and a half turns, the blood supply is cutoff entirely and the loop becomes gangrenous. The loop is instantly distended to its utmost capacity by gas (partly due to CO₂) which cannot be absorbed, and in a very short time bowel becomes cyanotic, water logged and gangrenous. A large quantity of blood gets sequestered and may cause fatal collapse. Death is usually due to peritonitis from perforation of the closed loop or from rupture of a gangrenous patch of intestine proximal to the twist itself.

CLINICAL FEATURES

There is often a history of acute attacks of left sided abdominal pain, probably due to partial volvulus, that untwists itself and is followed by the passage of large quantities of flatus and faeces.

Onset of volvulus is sudden and is characterized by severe abdominal pain, often coming on while the patient is straining at stool. Abdominal distension soon follows mainly left sided and in a matter of 6 hours the abdomen becomes distended. Hiccough and retching occurs early: vomiting is late. Constipation is absolute. Peristalsis is rarely visible in the distended loop. Marked pallor is often observed and due to shock and to loss of blood.

INVESTIGATIONS

- Plain x-ray abdomen erect reveals a distended sigmoid loop giving a coffee bean or bent inner tube appearance. The Frimann-dahl sign is pathognomonic and is often present. Haustral markings are absent. Three dense lines converging towards the obstruction are characteristic. Two air fluid levels are almost always seen within the sigmoid loop.
- Gastrograffin enema shows a narrowing at the site of torsion with “spiralling” of the mucosal folds and pathognomonic ‘birds beak’ or ‘ace of spades’

deformity and normal mucosal pattern in the rectum distal to the dilated loop.

Barium enema is contraindicated because of danger of perforation.

- CT scan is useful in sigmoid volvulus producing non specific pattern in plain X-ray. The “WHIRL SIGN” is the descriptive term for the CT appearance of volvulus. The whirl is constituted by afferent and efferent loop and central portion by

Twisted mesentery and bowel.

MANAGEMENT

- Operative detorsion
- Resection with primary end –to – end anastomosis
- Paul-mickulicz type double barrel colostomy
- Hartmann operation

Following resection , the surgeon has his choices, determined by local and general condition of the patient.

- Perform an end-to-end anastomosis to reconstitute colonic continuity. This is the preferred choice. The anastomosis should be in one layer, using interrupted inverting sutures o 2/0 or 3/0 non absorbable material.

- To bring both cut ends of bowel to the surface – the proximal ends as an end colostomy and the distal end separately as a mucous fistula – Paul Mickulicz operation and intestinal continuity is restored at later date.
- If the length of distal bowel available is not enough to bring it to the surface, the distal cut end is oversewn in two layers and returned to the pelvis, while the proximal cut end is brought out as an end colostomy – (HARTMANN’S procedure). Intestinal continuity is restored at a later date.

OBSTRUCTION CAUSED BY CAECAL VOLVULUS

Although volvulus of the caecum is the term commonly employed, it is anatomically misnomer, because the terminal ileum and ascending colon are usually involved. Other terms employed include volvulus of the ileocolic segment, ileocaecal volvulus and volvulus of the right colon.

The volvulus is nearly always in clockwise direction. The first twist obstructs the ascending colon: if a second twist occurs, it obstructs the ileum also.

PREDISPOSING FACTORS

- Abnormal mobility of both the caecum and the ascending colon occurs due to
- Complete malrotation
- A common ileocaecal mesentery

- Imperfect fixation of caecum
- Extremes of dietary intake
- Previous abdominal operations
- Post inflammatory peritoneal adhesions
- Upward displacement of caecum due to pregnancy, pelvic tumours cysts.

CLINICAL FEATURE

Occur in any age but majority in the 5th and 6th decades, it is about twice as common in females, caecal volvulus may manifest itself either as an acute or chronic obstruction.

The patient presents with colicky abdominal pain in the right lower quadrant with marked distension. The grossly dilated right colon producing a ‘Tympanitic’ mass situated in the right iliac fossa but flop over onto the left of the abdomen. Nausea, vomiting and absolute constipation are accompanying feature.

RADIOLOGICAL EXAMINATION

- Great distension of the caecum, often placed ectopically in the left upper quadrant

- Distended loops of small intestine located to the right of the caecal gas shadow.
- Visualization of the ileocaecal valve when the caecum is outlined by gas.
- Presence of a single fluid level in the caecum compared with two large fluid levels in case of sigmoid volvulus. Barium enema is most effective in diagnosis of chronic cases where opaque medium cut off at the transverse colon or at the hepatic flexure.

TREATMENT

- Detorsion
- Caecostomy – in cases with a small patch of gangrene on the caecal wall which was used as the site of caecostomy.
- Primary right hemicolectomy – when involved segment of intestine.

MALIGNANT OBSTRUCTION

Bowel obstruction and thereby bowel ischemia can even occur in primary or secondary neoplasms in abdomen. Common primary malignant condition includes colo-rectal, small bowel and ovarian neoplasms. Secondary neoplasms includes peritoneal carcinomatosis from gastric / pancreatic carcinomas and secondaries from malignant melanoma, breast, kidney and lung.

- 10 – 25 % patients with colorectal cancer and 20 – 50 % patients with ovarian cancer may present with intestinal obstruction at some point of time during the disease course.

- A setting of presentation involves a patient who has undergone surgery for malignant lesion in the past and now presents with intestinal obstruction either due to adhesions or recurrence. Recurrence is common among gastric and pancreatic neoplasms whereas adhesion is common in colorectal cancers.

Pathophysiology :

Bowel ischemia and necrosis proximal to the growth occurs by three ways.

- Bowel distention proximal to the obstruction.
 - Bacterial overgrowth due to stasis of bowel contents.
 - Direct mechanical compression of mesenteric vessels.
-
- Clinical features includes the signs and symptoms of obstruction according to the site of involvement.

Principle of management :

- Resection of gangrenous bowel and management of primary tumor accordingly or definitive palliative procedures.
- Whenever feasible, resection of tumour or bypass procedures should be done. While resecting the malignant lesion, enbloc resection of tumour , mesentery along with its lymphatics and the vascular pedicle is to be ligated at its origin.
- Since the bowel ischemia is often a terminal event in many of these patients and they are fit only for palliative procedures. So decision about the management if to do definitive or palliative procedures need to be individualised after weighing the risks. Benefits and life expectancy of the patient.
- several studies emphasized that palliation of obstruction can be achieved with surgery in 60 – 90 % patients with malignant obstruction.

MESENTRIC INFARCTION

Gut is normally protected from ischemia by its abundant collateral circulation but sudden occlusion at main branch or at more periphery beyond the largest collaterals is poorly tolerated and leads to bowel gangrene.

Causes :

Vaso occlusive diseases :

Thrombo – Embolism.

Non occlusive disease :

- vasculitis
- drugs
- shock

Patho physiology :

- Auto regulating vasodilatation failure due to increased perfusion pressure in ischemia.
- Over riding of vasoconstriction to that of vasodilation.

Clinical Features :

- mean age – 70 yrs.
- 2/3 rd of the patients have chronic heart disease / other vascular disease
- Acute abdominal pain out of the proportion to that of clinical sign is the classical picture.
- H/o vomiting in more than 1/2 of the patients.

- H/o diarrhoea in 1/3 rd of patient.
- Occult blood is positive in 25 % of cases.

Diagnosis :

Laboratory findings :

- Leucocytosis
- Elevated enzymes
- Serum lactate dehydrogenase
- Serum amylase
- Serum creatin phosphokinase
- Serum alkaline phosphatase
- Serum diamine oxidase.

Plain Xray Findings :

- multiple air fluid levels with edema of bowel wall
- Intra mural gas, air in portal vein is pathgonomic

CT abdomen findings :

- Thrombus / Embolism in mesenteric vessels.
- Intra mural air and air in portal vein

General consideration in MVD :

- Resection of necrosed bowel.
- Restore the blood flow – I) mesenteric vessels embelotomy

II) Bypass graft

- Regional (or) Systemic thrombolytic agents like streptokinase, urokinase and papaverine infusion.
 - Assess the viability of resected margins.
 - If viability is doubtful, II look surgery 12 – 24 hrs later, after adequate resuscitation.
 - Primary anastomosis is done only when no II look surgery is planned.
 - Anticoagulants like heparin in the post operative period.

SURGICAL STRATEGIES IN LARGE BOWEL INVOLVEMENT

- **For lesions in caecum and ascending colon :**

Procedure of choice is Right hemicolectomy. Usually with a primary anastomosis.

- **For lesion in the transverse colon :**

Extended right hemicolectomy with primary anastomosis with or without proximal diversion.

- **For lesion in the descending and sigmoid colon:**

- Left hemicolectomy : for descending colon growth
- Extended left hemicolectomy : for splenic flexure or sigmoid colon growth.
- Sigmoid colectomy : for mid sigmoid growths
- Subtotal colectomy : for left colon growths.

Followed by either primary anastomosis with or without diverting loop ileostomy or Hartmann's procedure / mucous fistula.

4) For lesions in rectum:

Primary resection in this situation has potential risk and therefore to be avoided.

If the patient is not a candidate for restoration of intestinal continuity, primary resection with end colostomy may be an acceptable approach.

Diversion procedures like sigmoid loop colostomy or transverse loop colostomy followed by definitive surgery in the future is the better choice.

Self expanding intraluminal stent (SEMS) is also useful when palliating patients who might not tolerate surgical diversion or those with unresectable disease and limited survival.

Materials & Methods

Materials

Study group :

50 cases with Bowel gangrene admitted under all surgical units at Government Thanjavur Medical College Hospital.

Inclusion Criteria

- Patients age group : 12 years & above
- Both sexes

Exclusion criteria

- Patient < 12 yrs

Total Number of cases studied

50 Cases

Study Period

May 2010 to November 2012

Data Collection regarding the study:

- Demographic details
- Time interval between onset of symptoms and surgery
- Clinical Presentation
- Imaging studies and Laboratory investigation
- Operative Findings & Cause for Gangrene

- Type of the treatment offered
- Eventual outcome of the patient.

Methodology

Study was a prospective observational study

Study group was 50 cases with Gangrenous Bowel disease admitted at Thanjavur Medical college Hospital, satisfying the inclusion & Exclusion criteria, during the period of 2 years from May 2010 to November 2012.

Detailed History, clinical Findings and Pre operative complications if any were noted.

All the patients were subjected to relevant radiological and Blood investigations with simultaneous resuscitation.

After adequate pre operative resuscitative measures that includes fluid & Electrolyte correction by intravenous route, naso gastric decompression and broad spectrum antibiotics, all the patients were subjected to Emergency surgical procedure appropriate to the condition.

Preoperative findings, intra operative complications if any and type of surgical procedure offered was documented.

Post operatively, all the patients were managed effectively under surgical intensive care unit with intravenous fluids, Antibiotics, etc.

Post operative follow up, complications if any and its management and final outcome of the patient were noted.

Various etiological factors leading to Bowel Gangrene, various mode of clinical presentation, various modalities of treatment and the factors which influence the morbidity & mortality of the patient with bowel gangrene were studied.

RESULTS

Results observed in our study of Bowel Gangrene in 50 patients were as follows: -

- SEX PREDILICTION: Total Number of Male
patients :35 (70%)

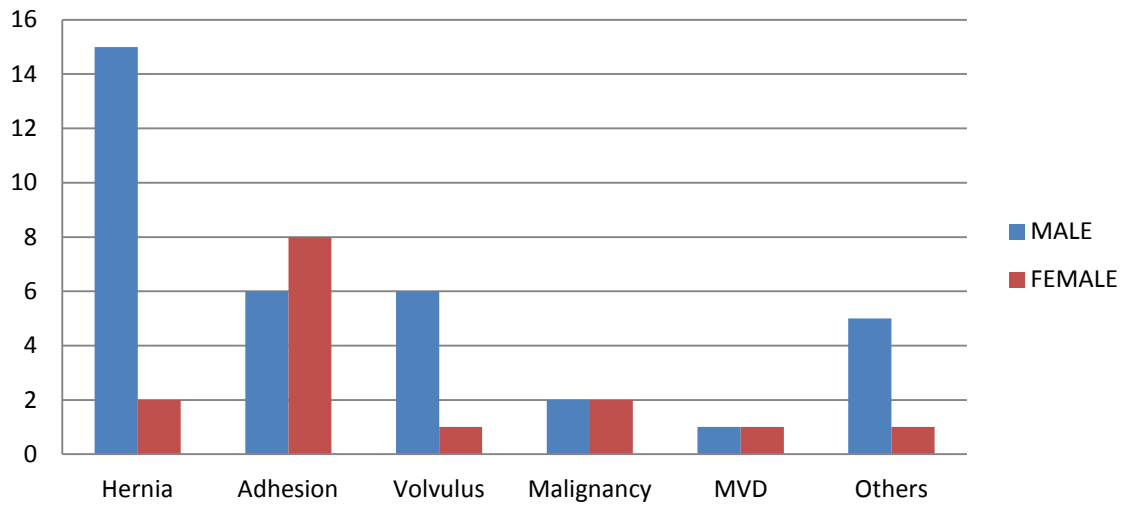
Female Patients: 15 (30%)

Table 1: Sex distribution among various causes

CAUSES	MALE	FEMALE
Hernia	15	2
Adhesion	6	8
Volvulus	6	1
Malignancy	2	2
MVD	1	1
Others	5	1
TOTAL	35	15

Among the individual causes, hernias are more common among the male patients and adhesions are more common among the female Population.

Sex distribution among various causes



- AGE DISTRIBUTION :

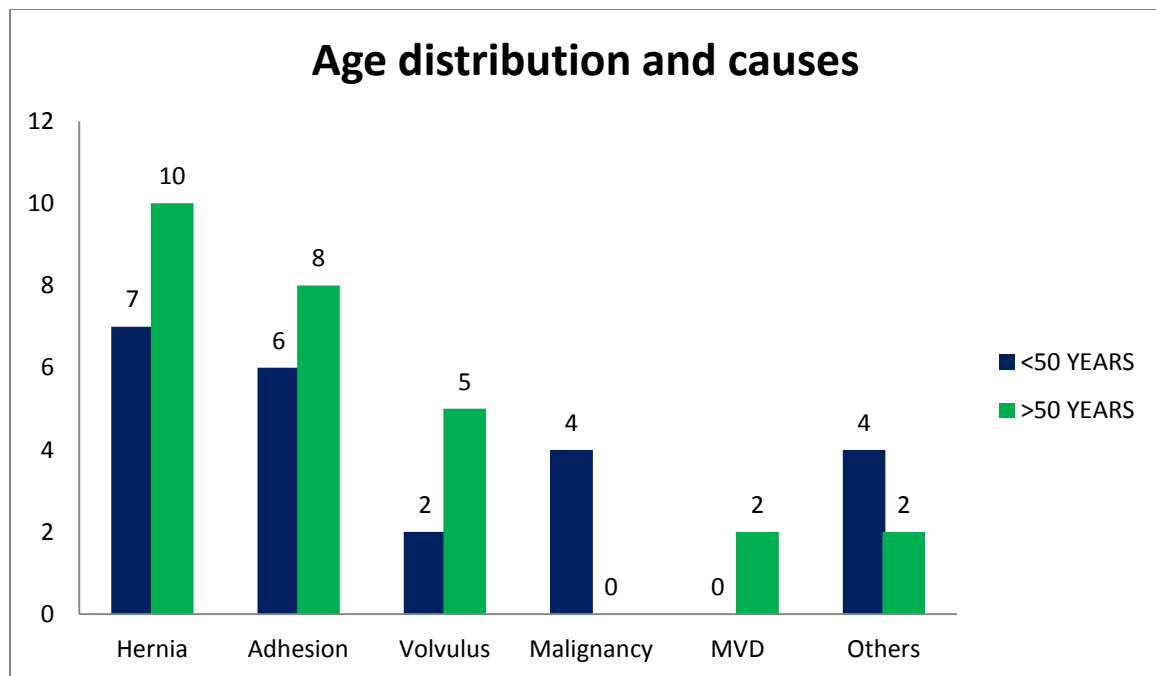
Total Number of Patients below 50 years of age group : 23 (46%)

Age above 50 years : 27 (54%)

Table 2 : Age distribution and causes

CAUSES	<50 YEARS	>50 YEARS
Hernia	7	10
Adhesion	6	8
Volvulus	2	5
Malignancy	4	0
MVD	0	2
Others	4	2
TOTAL	23	27

Among the individual diseases, Hernia, Volvulus and Mesenteric Vascular Diseases were common in Geriatric Population.



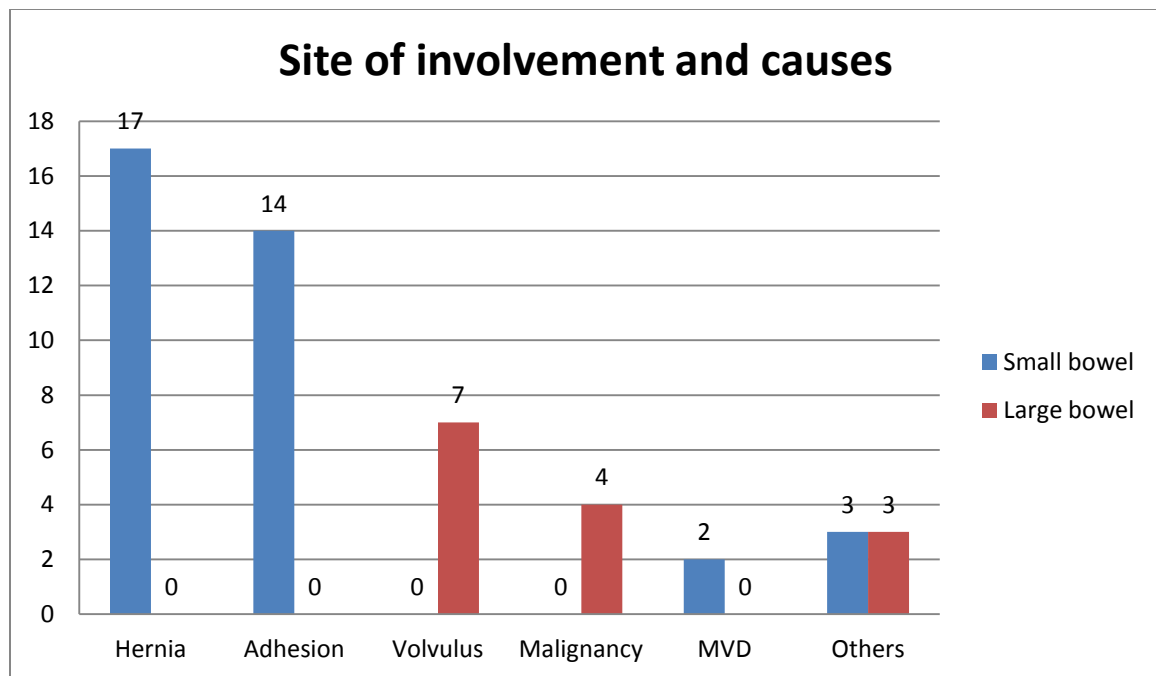
- SITE OF INVOLVEMENT :

Total Number of cases with small Bowel Gangrene : 36 (72%)

Total number of cases with large Bowel Gangrene : 14 (28%)

Table 3 : Site of involvement and causes

CAUSES	Small bowel	Large bowel
Hernia	17	0
Adhesion	14	0
Volvulus	-	7
Malignancy	-	4
MVD	2	-
Others	3	3
TOTAL	36	14



• **Table 4 :ETIOLOGICAL DISTRIBUTION**

CAUSES	NO.	PERCENTAGE
Hernia	17	34
Adhesion	14	28
Volvulus	7	14
Malignancy	4	8
MVD	2	4
Others	6	12
TOTAL	50	14

In our study, still Hernia was the leading cause of Bowel Strangulation, (34%) followed by Adhesive obstruction and Volvulus (14% to 28%)

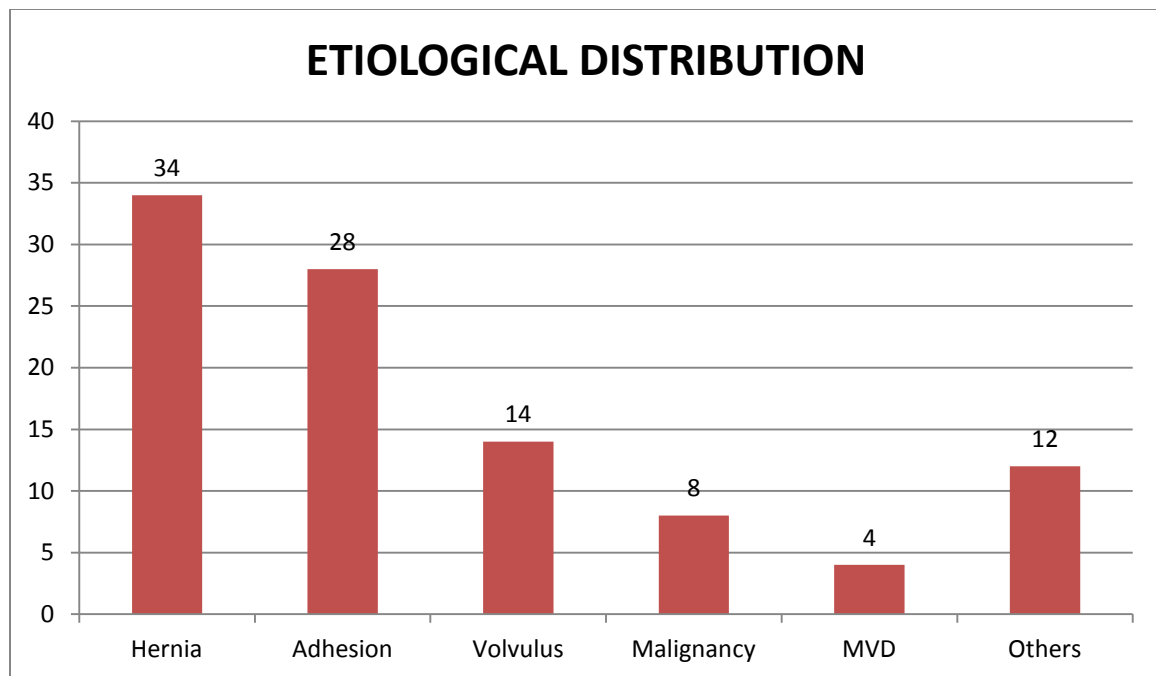


Table 5 : Types of hernias

TYPES OF HERNIA	NO.	PERCENTAGE
Inguinal Hernia	15	88
Incisional Hernia	1	11
Femoral Hernia	1	11

Among the Hernia Inguinal Hernia contributed 88%

Table 6 : Types of adhesions

ADHESIONS	NO.	PERCENTAGE
Post inflammatory	9	65
Post Surgical	5	35

Among the adhesive obstruction, Post inflammatory obstruction contributed to 65% (Numbers. 9) and Post surgical obstruction contributed to 35% (Numbers. 5)

MORTALITY :

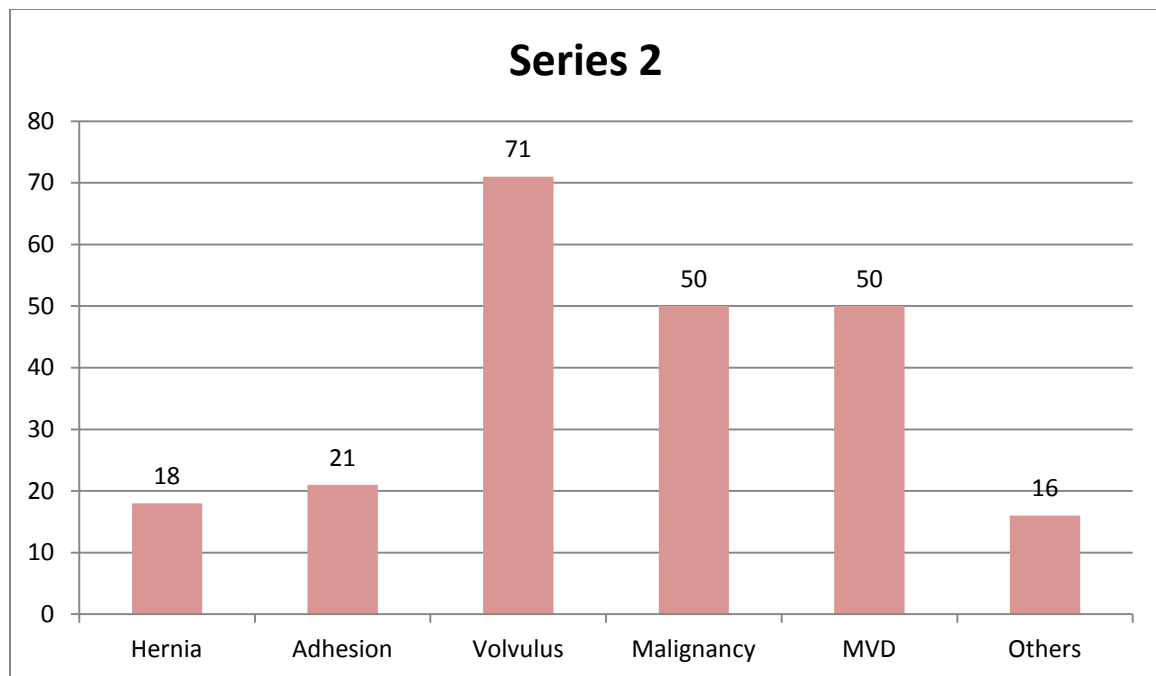
Overall Mortality Rate in our study is 30% (Numbers. 15)

Distribution of mortality according to underlying causes.

Table 7 : Cause related mortality

CAUSES	NO.	PERCENTAGE
Hernia	3	18
Adhesion	3	21
Volvulus	5	71
Malignancy	2	50
MVD	1	50
Others	1	16
TOTAL	15	30

Among the individual causes, Volvulus had a high mortality rate of 71% followed by Mesenteric Vascular disease and Malignancy, both of which had 50% mortality rate. Mortality Rate among Hernia was 18% (Numbers. 3) and Adhesion was 21% (Numbers. 3)



• **Table 9 : AGE RELATED MORTALITY:**

CAUSES	BELOW 50 YEARS			ABOVE 50 YEARS		
	Cases	No.	Per	Cases	No.	Per
Hernia	7	1	14	10	2	20
Adhesion	6	1	17	8	2	25
Volvulus	2	1	50	5	4	80
Malignancy	4	2	50	0	0	0
MVD	0	0	0	2	1	50
Others	4	1	25	2	0	0
TOTAL	23	6	26	27	9	33

Overall Mortality Rate among the age group below 50 years: 26% (No.6)

Mortality Rate among the age group above 50 years : 33% (No.9)

Overall the mortality rate increased with age regardless of etiology

Table 10 : MORTALITY RATE RELATED TO DURATION OF SYMPTOMS:

CAUSES	<36 hours			>36 hours		
	Cases	No.	Per	Cases	No.	Per
Hernia	7	0	0	10	3	30
Adhesion	8	1	13	6	2	33
Volvulus	5	3	60	2	2	100
Malignancy	0	0	0	4	2	50
MVD	2	1	50	0	0	0
Others	3	1	33	3	0	0
TOTAL	25	6	24	25	9	36

Overall Mortality Rate found to be increased with duration of symptoms regardless of etiology

Mortality Rate when presented within 36 hours of onset : 24%

Mortality Rate when presented after 36 hours of onset of symptom: 36%

Table 11 : Mortality Rate related to site of Bowel

involvement

Site	Case	No. of death	Percentage
Small Bowel	36	7	19
Large Bowel	14	8	57

Mortality rate with Small Bowel Gangrene was 19% (Numbers. 7) and that of Large Bowel Gangrene was 57% (Numbers. 8)

Table 12 : MORTALITY RELATED TO LENGTH OF BOWEL INVOLVEMENT:

CAUSES	<100CM			>100CM		
	Cases	No.	Per	Cases	No.	Per
Hernia	14	3	21	3	0	0
Adhesion	7	1	14	7	2	29
Volvulus	0	0	0	7	5	71
Malignancy	0	0	0	4	2	50
MVD	0	0	0	2	1	50
Others	2	0	0	4	1	25
TOTAL	23	4	17	27	11	41

Overall the mortality rate found to be increased with the length of Bowel involvement.

Mortality rate when the length of Bowel resected below 100 cm 17%

Mortality rate when the length of Bowel resected above 100 cm 41%

Table 13: MORTALITY RELATED TO PREOPERATIVE MORBID CONDITIONS:

CAUSES	HYPOVOLEMIC SHOCK			PERITONITIS			SYSTEMIC ILLNESS		
	Cases	No.	Per	Cases	No.	Per	Cases	No.	Per
Hernia	2	1	50	1	1	100	5	1	20
Adhesion	3	0	0	4	2	50	10	1	10
Volvulus	2	1	50	3	3	100	2	1	50
Malignancy	1	0	0	3	1	33	0	0	0
MVD	1	1	100	0	0	0	1	0	0
Others	1	1	100	0	0	0	1	0	0
TOTAL	10	4	40	11	7	64	20	4	20

Mortality Rate associated to Hypovolemic shock : 40%

Peritonitis : 64%

Systemic illness : 20%

Table 14 : MORTALITY RATE RELATED TO PRESENCE OF PERFORATION AND FECAL PERITONITIS

	CASES	DEATH	PERCENTAGE
Perforation	6	4	67
Fecal Peritonitis	3	3	100

Fecal Peritonitis carried the risk of 100% mortality, while perforation carried the risk of 67% mortality.

5. (H). IMMEDIATE CAUSE FOR DEATH.

Among the 15 patients who were died.

- 7 Patients were died due to septicemia shock.
- 2 Patients were died due to Progressive renal insufficiency
- 2 Patients were died due to failure to wean from ventilator.
- 2 Patients were died due to ARDS.
- 2 Patients were died due to Cardiogenic shock.

Table 15 :INCIDENCE OF POST OPERATIVE COMPLICATION: -

COMPLICATION	NO.	PERCENTAGE
Anastamotic Leak	2	4
Local sepsis	5	10
Systemic Complications	3	6
TOTAL	10	20

Overall Percentage of Morbidity : 20%

Incidence of Anastamotic Leak after surgery : 4% (No.2)

Local Sepsis after Surgery : 10% (No.2)

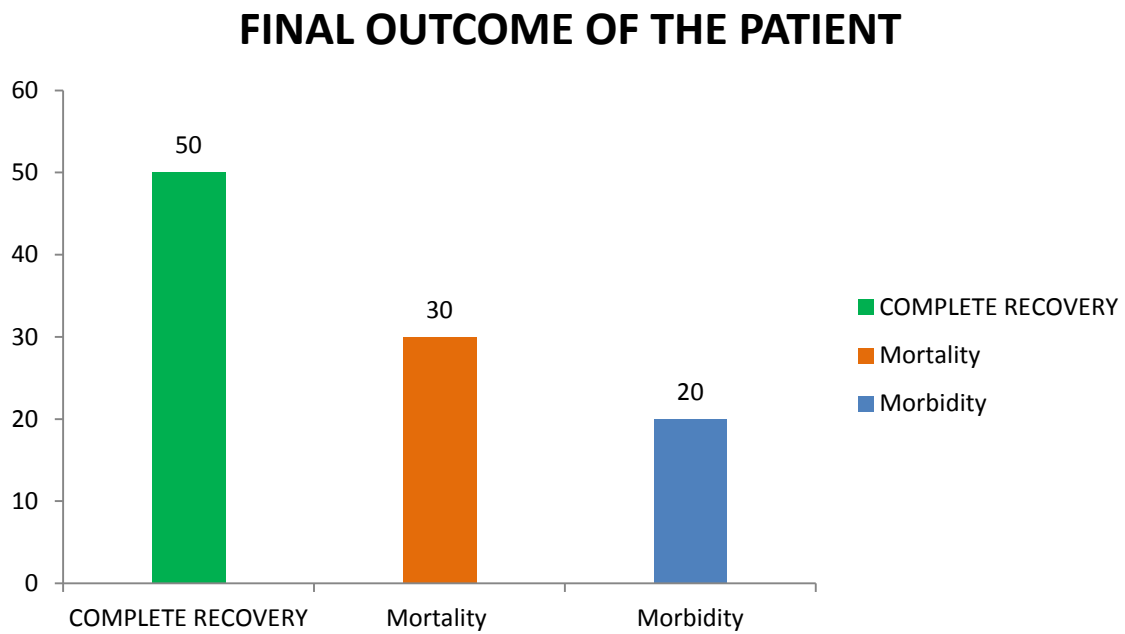
Systemic Complication : 9% (No.3)

- **FINAL OUTCOME OF THE PATIENT :**

Complete Recovery : 50%

Mortality : 30%

Morbidity : 20%



DISCUSSION

Bowel Gangrene is a complex disorder with various primary causes and clinical presentations. Acute Vascular compromise either due to intestinal obstruction or the due to mesenteric vascular diseases is the prime cause for Bowel Gangrene. Over the year, mortality and morbidity among the patients with simple intestinal obstruction and mesenteric vascular disease have been come down. But equivalent degree of improvement has not been attained where Gangrenous Bowel complicated the situation. The final outcome of the patient with bowel Gangrene is influenced by variety of factors like underlying Etiology, Severity of illness, Functional status of patient at presentation, presence of complications like shock, perforation / fecal peritonitis and prompt and effective resuscitation and management.

The main aim of our study is to analyses the various causes behind the bowel gangrene, to evaluate the final outcome of the treatment offered and to enumerate various factors influencing the morbidity and mortality associated with Bowel Gangrene.

- The total number of patients included in our study were 50
- Male constituted 70% (Numbers . 35) and
Female constituted 30% (Numbers. 15)

The most frequent cause for Bowel Gangrene in our study was strangulated inguinal Hernia and hence male Predominance.

- In our study incidence of Bowel Gangrene found to be increased with age advancement.

Total number of patients with Bowel Gangrene below the age of 50 years was 23; contributing 46% of total and above the age of 50 years were 27 contributing for 54 % of Total.

It is explained by increased prevalence of Mesenteric Vascular disease, Malignancy and Hernia among the old age group.

- The site of the lesion was found to be small bowel in vast majority 72% (Numbers. 36)

Large Bowel contributed to 28% (Numbers. 14%)

This is because,

- Prevalence of colonic Neoplasm and colonic diverticuli are low in our population.

(2)Colonic Ischemia resulting from obstruction / vascular occlusion is quite a chronic process than acute one and hence the incidence of gangrene in large bowel is relatively less.

5) In our study, the frequent cause of Bowel Gangrene were Hernia 34% (numbers. 17) Adhesions 28% (Numbers. 14) Volvulus 14% (Numbers. 7) only 2 cases of MVD were noted in our study, Which contributed only about 4%. The rest were malignancy 8% (Numbers. 4) Miscellaneous Conditions like intussusceptions, Electrical Shock and Stricture Contributed 12% (Numbers. 6).

Among the Hernia, Inguinal Hernia contributed about 88% (15 cases) in our study. Only one case of strangulated Femoral Hernia and one case of strangulated incisional hernia were recorded which contributed 11% each.

High prevalence of Hernia causing strangulation and thereby causing bowel gangrene in our part is partly attributed to reluctant of the patient for elective surgical repair of hernia due to poverty, ignorance and general fear of surgery and partly due to increased prevalence to chronic respiratory diseases like Tuberculosis in our population.

Coming to adhesive obstruction, Post inflammatory adhesion contributed about 66% (Numbers. 9) and Post surgical adhesion contributed about 35% (Numbers. 5). Increased Prevalence of chronic inflammatory disorders like Tuberculosis, Pelvic inflammatory diseases and increase in the number of laparotomies has raised the incidence of adhesive obstruction.

(5) Overall mortality rate in our study was 30% (Numbers. 15)

(5)(a) Among the various causes of Bowel strangulation sigmoid volvulus had high mortality rate about 71% (Numbers. 5). It is mainly due to early development of peritonitis / Fecal contamination as a result of closed loop obstruction.

Old age and other co. morbid conditions like CAHD, DM associated with MVD are responsible for high mortality (50%) in patients with bowel gangrene due to MVD.

Hernia was the leading cause for bowel gangrene in our study. But Mortality rate among the patients with hernia was found to be low – 18% (Numbers. 3)

This is because,

- Early presentation of patient
- Early Recognition and Treatment
- Lethal Toxins confined to hernia sac

(5)(b) Mortality rate was found to be increase with age due to age related complications with respect to disease, surgery and anesthesia.

- (5)(c) Mortality Rate observed among 25 cases who were operated upon within 36 hours from the onset of symptoms was 24% and after 36 hours was 36%. Complications like shock, Peritonitis found to be increased with delay in presentation / Management and hence the mortality.
- (5)(d) Large Bowel Gangrene contributed to high mortality (57%) when compared to small Bowel Gangrene (19%) and this is because of increased incidence of fecal peritonitis in large bowel involvement.
- (5)(e) When the length of bowel affected / resected exceeded 100 cm, the mortality rate increased from 17% to 41%. It is naturally due to extent of disease and sepsis.
- (5)(f) Preoperative morbid conditions like peritonitis, shock and other comorbid conditions like DM, CAHD, Renal insufficiency, all found to have a profound influence over the outcome of the patient.

Mortality Rate associated with Hypovolemic Shock - 40% (Numbers.4)

Mortality Rate associated with Peritonitis - 47% (Numbers.7)

With systemic illness, mortality rate was only 20%

Thus local complication like perforation and fecal peritonitis found to have greater influence than co-morbid systemic illness.

(6) Post Operative complications observed were.

Anastomotic leak in 2 Patients, local sepsis in 5 patients and systemic complications in 3 patients which include progressive renal insufficiency, pneumonia and septicemia.

(7) Final outcome of the patient with Bowel Gangrene observed were Complete recovery achieved in 25 patients (50%) 15 patients were died and rest of the patients were recovered but with some sort of complications as mentioned earlier.

And thus, in our study, mortality and morbidity associated with Bowel Gangrene were 30% and 20% respectively. 50% of the patients with bowel gangrene recovered completely without any complication.

CONCLUSION

- Intestinal strangulation from hernia, adhesions and Volvulus was the major cause for Bowel Gangrene. Mesenteric Vascular diseases contribute only about 4% in our study.

(1)(a) Among the Bowel strangulation, Hernia is leading cause for bowel gangrene in our study (34%) of which Inguinal Hernia alone accounts for 88% in total

(b) Strangulation following adhesive obstruction comes as the 2nd leading cause for Bowel Gangrene (28%) of which post inflammatory adhesions is the major cause (65%).

(c) Sigmoid Volvulus ranks 3rd in etiology of Bowel Gangrene contributing 14%. Large Bowel Neoplasm contributes 8%.

(2) The final outcome of the treatment offered is as follows: -

(a) Complete recovery is attained in 50% of patients.

- (b) Overall mortality rate is 30%. This figure is 18% for hernia, 21% for Adhesions 71% for sigmoid volvulus and 50% for malignancy and 50% for MVD.
- (c) Septicemic shock is the prime cause of death in vast majority of patients with Bowel Gangrene (47%). Failures to wean from ventilator, ARDS, Progressive renal insufficiency and cardiogenic shock together contribute the rest.
- (d) Overall morbidity rate is 20%. Among these local complications like Anastomotic leak and local sepsis together constitute about 14%.
- (3) Old age, premorbid illness, treatment delay, and Fecal contamination are significantly associated with increased morbidity and mortality rate.
- (4) Mortality and Morbidity associated with Bowel Gangrene can be limited by following measures:
 - (a) Pre operative optimization of hemodynamic and respiratory parameters.
 - (b) Control the sepsis with liberal use of antibiotics.
 - (c) Early surgical intervention and removal of devitalized Bowel, before lethal toxins enters into the circulation.
 - (d) Careful planning and Technique during definitive surgical therapy.
 - (e) Recognition of Barriers to healing and control over them.

BIBLIOGRAPHY

Alfred Cuschieri, disorders of abdominal wall and peritoneal cavity, intestinal obstruction in Sir Alfred Cushchieri's Essential Surgical Practise" fourth edition, page 156-162

Bizer LS, Liebling RW, Delaney HM : Small bowel Obstruction. Surgery 89: 407-413, 1981.

Brolin RE; The role of GI tube decompression in the treatment of Mechanical intestinal obstruction. Am. Surgeon 49: 131-137, 1983

Bruce G. Wolff and Anne Mary Boller, Large bowel obstruction, Acute colonic pseudo obstruction, colonic volvulus in current surgical theraphy by John L. Cameron, ninth edition, page 189-197.

Das, P. Shukla HS: Clinical diagnosis of Abdominal Tuberculosis Brit. J. Surg 63: 941, 1976.

Danekar GW, Carlson GW, Hohn DC, Lynch P, Levin B: Endoscopic Laser Recanalization is effective for prevention and treatment of obstruction in sigmoid and rectal cancer. Arch. Surg. 1991 Nov. 126 (11) : 1348-52

Edward E. Whang, Stanley W.Ashley and Micheal J. Zinner, small bowel obstruction in Schwartz's principle of surgery, eighth edition, page 1027-1032

Kelly M. Bullard and David A. Rothenberger, Volvulus of sigmoid, caecal volvulus, colonic pseudo obstruction, ostomies, resections, therapy for rectal and colonic carcinoma in Schwartz's principle of surgery, eighth edition, page 1055-1117

Kurtis A Campbell , Small bowel Obstruction in current Surgical Theraphy by john L. Cameron, ninth edition, page 117-120

Goar lav, Barmiia NN, Domanski BV, Esebua BA: Surgical treatment of acute intestinal obstruction. Klin Khir 1991(4): 1-3

Harlow CL, Stears RL, Archer PG: Diagnosis of bowel obstruction on plain abdominal radiographs: Significance of air-fluid level at different heights in the same loop of bowel AJR Am.J. Roentgenol 1993. Aug.161(2): 291-5.

Harlod Ellis in Maingot's Abdominal Operations, 10th edition, P.1159-1172, 1391-1402

Iko Bo, Teal JS, Siran SM: Computed Tomography of adult intususception clinical and experimental studies, AJR 143: 769-772, 1984.

Lelcuk S, Klauser JM, Merhan A. Endoscopic decompression of acute colonic obstruction. Ann. Surg. 203: 292-294, 1986.

Liu MY, Lin HH, Wu CS, JanYY , Wang KL: etiology of Intestinal Obstruction - \$ years experience. Chang Kang I H such 1990 sep. 13(3): 161-6

Lund L, Gandrup, Baslev I: Colonic Ileus – Treatment of colonic ileus, a 10 year patient material, Ugeskr Laeger 1993 Apr.26: 155 (17): 1284-4

Mandeep Saund and David I. Soyvel, bowel obstruction in Greenfield's surgery" scientific principles and practice" fourth edition 767-787

Marc Christopher Winslet, Intestinal Obstruction in Bailey and Love's short Practice of Surgery 25th edition, page 1188-1203

Mark Evers B, Small intestine obstruction in Sabiston's Textbook of Surgery 18th edition, page 1289-1297

Munro. A.A Intestinal obstruction in Hamilton Bailey's Emergency Surgery, 13th edition, pg 420-445

Pujari B.D, Abdominal Tuberculosis in The ASI textbook of Surgery, 2003, pg 501-503

Rebekah R. White and Danny O. Jacobs, Small bowel Volvulus in Shackelford's surgery of the Alimentary Tract, 6th edition, pg 1035-1041

Robert J. Fitzgibbons, Jr. Charles J. Filipi and Thomas H.Quinn, Hernia accident(incarceration, bowel obstruction, strangulation), groin hernias in women. Schwartz's Principle of Surgery, 8th edition edition, pg 1355-1358, 1390.

Scott G. Houghton, Antonio Ramos De La Madena Ardlm, Michael G.Sarr, Bowel Obstruction in Maingot's Abdominal Operations 11th edition: pg 479-508

Scott Helton W and Piero M Fisichella, Intestinal Obstruction in ACS surgery 6th edition, pg514-533

Simon Paterson- Brown, Benjamin N.J Thomson, in intestinal obstruction, in The New Airds Companion in surgical studies, 3rd edition, pg 585-590.

Soo Y.Kin and Jon B. Morris, Small Bowel obstruction, in Shackelford's Surgery of the alimentary Tract, 6th edition, pg 1025-1034

Tehemton E. Udwadia and Rushad T. Udwadia, small bowel obstruction, The ASI Text book of Surgery, 2003 pg 443-448

Thomas A.Miller and Jeannie F. Savas, Post Gastrectomy syndromes in Shackelford's surgery of the alimentary tract, 6th edition,pg 879,880

Binns JC.Issaccson P. Age-related changes in the colonic blood supply : their relevance to ischaemic colitis. Gut 1978;76;239.

Brandt I.J. Boley SJ.Colonic ischemia. Surg Clin North Am 1992;72:203

Barnett, W. O. Trunett, G., Williams, R. And Crowell, J.: Shock in strangulation Obstruction : Mechanisms and Management . Ann. Surg., 157:747, 1963.

Skinner, D. B., Zarins, C. K. and Mossa, A. R.: Mesenteric Vascular Disease, Am. J. Sur., 128:835, 1974.

Peroperative Picture of Large Bowel Gangrene



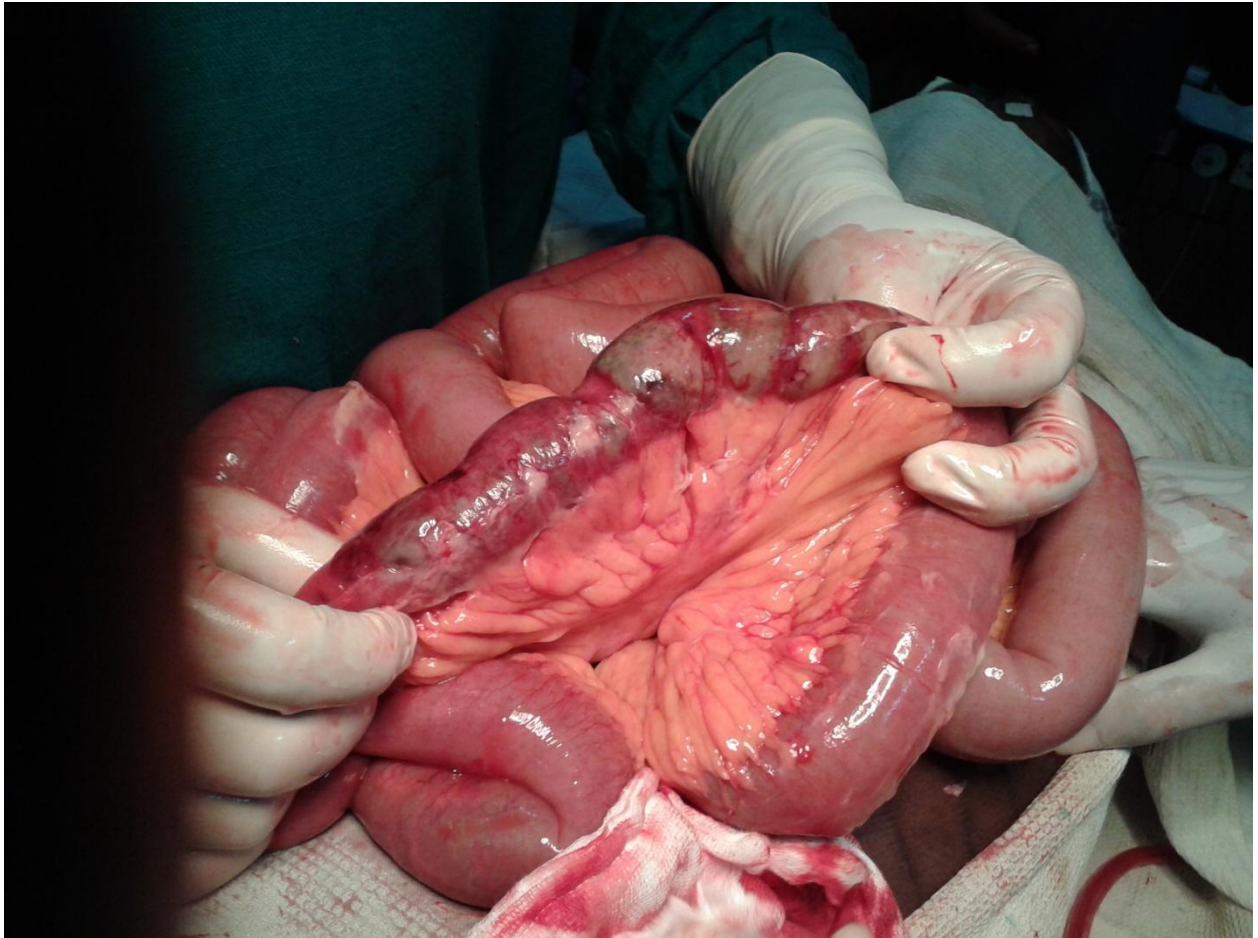
X- Ray Picture of Large Bowel Obstruction



Post Operative Picture of Large Bowel Gangrene



Peroperative Picture of Patchy gangrene of Small Bowel with perforation



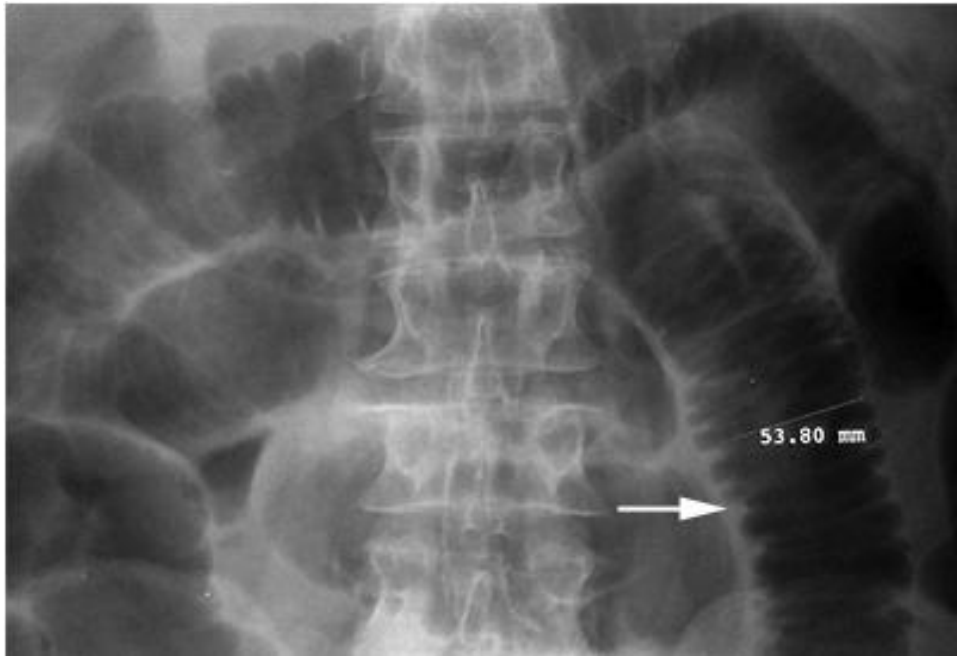
Pre –Operative Picture of Strangulated Femoral Hernia



Primary Anastomosis of Small Bowel in Strangulated Hernia



X- Ray Picture of Small Bowel Obstruction



No.	Name	Aged Sex	ID No.	Cause	Duration of Symptoms		Pre morbid illness			Per operative Findings				Procedure	Final Outcome			
							Shock	Peritonitis	Others	Site	Length		Perforation	Fecal Peritonitis		Complete Recovery	Death	Complications
					<36 hr	>36 hrs.					<100 cm	>100 cm						
1.	Singaram	45/M	1337938	SIH(L)	?	-	-	-	PRF	SB	-	?	-	-	Resection Anastamosis Hernia & Repair	?	-	-
2.	Thanseela	57/F	1337429	Adhesions	?	-	?	-	-	SB	?	-	-	-	Resection, Anastmosis & Adhesion Release	?	-	-
3.	Chellamuthu	71/M	1339148	SIH(L)	-	?			PRF	SB	?	-	-		Resection Anastamosis Hernia & Repair	-	-	Progressive Renal Insufficiency
4.	Kaliamoorthy	40/M	1342794	Hepatic Flexure Growth	-	?	?	-	-	LB	-	?	-	-	® Hemicolectomy Ileo – transverse Anastamosis	-	-	Wound Infection
5.	Thangavel	63/M	1345313	SIH(L)	-	?			COPD	SB	-	?	-	-	Resection + Anastamosis Hernia Repair	-	?	-
6.	Palanivel	45/M	1357611	Electrical Burns	-	?	-	-	-	LB	-	?	?	-	® Hemicolectomy Ileo – transverse Anastamosis	-	?	-
7.	Varadharajan	45/M	1363209	Sigmoid Volvulus	-	?	?	-	COPD	LB	-	?	-	?	Resection & Hartmann’s Procedure	-	?	-
8.	Krishnamoorthy	40/M	1364662	SIH®	-	?	?	-	-	SB	?	-	-	-	Resection & Anastamosis	?	-	-
9.	John	20/M	1365838	SIH®	?	-	-	-	COPD	SB	?	-	-	-	Resection & Anastamosis	?	-	-

10.	Samikannu	57/M	1347008	Band	-	?	-	?	-	SB	-	?	?	-	Resection & Anastomosis Band division	-	?	-
11.	Vasanth	60/F	1368070	Sigmoid Volvulus	-	?	-	?	PRF	LB	-	?	-	-	Hartmann's Procedure	-	?	-
12.	Ramalingam	50/M	1374790	Sigmoid Volvulus	-	?	?	-	-	LB	-	?	-	?	Hartmann's Procedure	-	?	-
13.	Paramasivam	43/M	1375697	Growth in Cecum	-	?	-		-	LB	-	?	-	-	® Hemicolecotomy & Ileo – transverse Anasotamosis	-	-	Delay in Recovery from Anesthesia
14.	Velayutham	55/M	1376076	SIH(L)	-	?		-	-	SB	-	-	-	-	Resection, Anastomosis Hernia Repair	?	-	-
15.	Jayakumar	45/M	1379486	Ileo colic Intussuption	?	-	-	-	DM	LB	-	-	-	-	Resection, Ileotransverse Anastomosis	-	-	Anastamotic Leak
16.	Karuppiah	60/M	1389081	Sigmoid Volvulus	-	?		?	-	LB	-	?			Resection, Hartmann's Procedure			
17.	Ponnian	50/M	1384584	Ileo-sigmoid Knotting	?	-	-	-	-	SB+ LB		?	-	-	Resection Ileo – transverse Anastomosis Distal descending loop colostomy	-	-	Intra abdominal abcess
18.	Meenaxi	70/F	1359881	Strangulated Femoral Hernia	-	?	-	?	CAHD	SB	?	-	-	-	Resection Anastomosis Hernia Repair	-	?	-
19.	Thirugnana m	57/M	1392796	SIH®	-	?	-	-	-	SB	?	-	-	-	Resection Anastomosis Hernia Repair	?	-	-
20.	Ramasamy	18/M	1396941	Sigmoid Volvulus	-	?	-	?	HT	LB	-	?	-	?	Resection, Hartmann's Procedure	-	?	-

21.	Maniarasu	18/M	1400986	SIH®	?	-	-	-	-	SB	?	-	-	-	Resection Anastomosis Hernia Repair	?	-	-
22.	Murugesan	22/M	140278	Ileo – colic Intussuc eption	?	-	-	-	PRF	SB	-	-	-	-	® Ileo – transverse Anastomosis	?	-	-
23.	Murugesan	70/M	1403837	Sigmoid Volvulus	?	-	?	-	-	LB	-	?		-	Resection & Anastomosis	-	-	Pneum onia
24.	Karthikeya n	42/M	1402915	Adhesio n	-	?	?	-	-	SB	?	-	-	-	Resection Anastomosis Adhesion Release	?	-	-
25.	Soundaraja n	60/M	1403631	SPH®	?	-	-	-	PRF	SB	?	-	-	-	Resection Anastomosis Hernia Repair	?	-	-
26.	Marudhapp an	60/M	1404182	SIH(L)	-	?	-	-	-	SB	?	-	-	-	Resection Anastomosis Hernia Repair	?	-	-
27.	Ramasamy	60/M	1406264	SIH®	-	?	-	-	CAHD	SB		?	-	-	Resection Anastomosis Hernia Repair	-	?	-
28.	Paramasiva m	53/M	1409116	SIH(L)	-	?	-	-	-	SB	?	-	-	-	Resection Anastomosis Hernia Repair	?	-	-
29.	Vellammal	48/F	1410684	Adhesio ns	?	-	-	-	COPD	SB	?	-	-	-	Resection & Anastomosis	?	-	-
30.	Ramu	40/F	1402318	Growth Rectum	-	?	-	?	-	LB		?	-	-	Resection, Double barrel sigmoid colostomy		?	-
31.	Ramkumar	23/M	1407420	Adhesio n	?	-	?	-	DM	SB	?	-	-	-	Resection, Anastomosis Adhesion Release	?	-	-
32.	Samiamma	43/F	1413266	Adhesio n	-	?		-	-	SB	?	-	-	-	Resection, Anastomosis Adhesion Release	?	-	-

33.	Marikannu	47/F	1415292	Band	?	-	?		-	SB	?	-	?		Resection Omental Band Release, Anastomosis	?	-	-
34.	Vaduvammal	60/F	1414131	Band	-	?	-	-	-	SB+ LB	?		?		Resection, Anastomosis, Transverse, loop colostomy	-	?	-
35.	Saraswathi	45/F	1415063	Growth Rectum	?	-	-	?	-	LB	?	-	-		Resection Double Barel sigmoid colostomy	-	?	-
36.	Thilainayaki	50/F	1416953	Sigmoid Volvulus	-	?	-	?	-	LB	-	-	-	-	Resection, Double Barel Sigmoid colostomy	-	?	-
37.	Gnanamal	50/F	1418132	Strangulated Inusional Hernia	?	-	-	?	-	SB	-	-	-	-	Resection, Anastomosis Herina Repair	?	-	-
38.	Ramar	61/M	1417866	MVD	?	-	-	-	CAHD	SB	-	-	-	-	Jeylino Transverse Anastomosis, with distal ileal ilosure	?	-	-
39.	Maniyan	65/M	1420406	Band	-	?	-	?	-	SB	?	-	?	-	Resection, Jeyuno, Transverse Anastomosis	-	?	-
40.	Govindaraj	42/M	1403535	Multiple Jeynnal stricture	-	?	-	-	DRF	SB	-	?	-	-	Resection, Jeyuno – Jeyunal Anastomosis	-	-	Burst Abdomen
41.	Kaliya Perumal	60/M	1403805	Stricture cecum	-	?	-	-	PD	LB		?	?		Resection ileo – transverse Anastomosis	-	-	Anastomosis
42.	Fransis	65/M	11431272	Adhesion	?	-	-	-	CAHD	SB	?	-	-	-	Resection, Anastomosis Adhesion Release	?	-	-
43.	Rengasamy	57/M	1421972	Ileal stricture	-	?	-	-	PT	SB	?	-	-	-	Resection and Jejuno – Transverse Anastomosis	?	-	-
44.	Sushila	60/F	1416755	MVD	?	-	?	-	-	SB		?	-	-	Jejuno – Transverse Anastomosis	-	?	-

45.	Sumathy	54/F	1403553	Adhesio n	?	-	-	-	PRF	SB	?	-	-	-	Adhesion Release Resection, Anastomosis	?	-	-
46.	Raja	45/M	1413696	SIH(L)	?	-	-	-	-	SB	?	-	-	-	Resection, Anastomosis Hernia Repair	?	-	-
47	Adaikalam	48/F	1402220	Adhesio n	?	-	-	-	-	SB	?	-	-	-	Resection, Adhesion Release, Anastomosis	?	-	-
48.	Padmanath an	58/M	1401154	SIH®	-	?	-	-	PRF	SB	?	-	-	-	Resection, Anastomosis Hernia Repair	?	-	-
49.	Palanimuth u	61/M	1409077	SIH ®	?	-	-	-	-	SB	?	-	-	-	Resection, Anastomosis Hernia Repair	?	-	-
50.	Muniamma l	59/F	1499921	Adhesio n	?	-	-	-	-	SB	?	-	-	-	Adhesion Release, Resection, Anastomosis	?	-	-

PATIENT PROFORMA												
PATIENT DETAILS	NAME											
	AGE											
	SEX											
	I.P.No.											
	OCCUPATION											
	PLACE											
	DATE OF ADMISSION											
	DATE OF SURGERY											
	DATE OF DISCHARGE											
Time interval between onset of symptoms and surgery												
PRESENTING SYMPTOMS	PAIN ABDOOMEN			VOMITING		NON - BILIOUS						
	MODE OF ONSET					BILIOUS						
	SITE OF PAIN	AT ONSET				FECULENT						
		AT PRESENT				FREQUENCY						
		RADIATION		ABDOMINAL DISTENSION	YES		NO					
				PASSED MOTION	YES		NO					
	DURATION											
	TYPE	INTERMITTENT		PASSED FLATUS	YES		NO					
		STEADY		BLEEDING PER RECTUM	YES		NO					
		COLICKY		FEVER	YES		NO					
	SEVERITY	MODERATE		DECREASED URINE OUTPUT	YES		NO					
		SEVERE		OTHERS								
	AGGRAVATING FACTOR											
	RELEIVING FACTORS											
	CO – MORBID ILLNESS	DM		BLEEDING DISORDER		DRUGS						
HT		EPILEPSY		PREVIOUS ABDOMINAL SURGERY								
COPD		JAUNDICE										
TB		ALCOHOL										
CHRONIC CONSTIPATION		SMOKER										

CLINICAL FINDINGS	GENERAL EXAMINATION		SYSTEMIC EXAMINATION				
	NORUISHMENT		ABDOOMEN				
		POOR BUILT	DISTENSION	YES		NO	
		MODERATELY BUILT					
		WELL BUILT		CENTRAL			
	MOOD			GENERALISED			
		NORMAL	GUARDING	YES		NO	
		UPSET					
		ANXIOUS	RIGIDITY	YES		NO	
	VITALS						
		TEMPARATURE	FREE FLUID	YES		NO	
		BLOOD PRESSURE					
		PULSE RATE	BOWEL SOUNDS	NORMAL			
	RESPRIATORY RATE						
	DEHYDRATION			ABSENT			
		MILD		INCREASED			
		MODERATE					
		SEVERE	PERISTALISIS	YES		NO	
	COLOUR		SCARS	YES		NO	
		NORMAL					
		PALLOR	MASS	YES		NO	
		JAUNDICED					
	CYANOSED	HERNIAL SITES					
	CVS EXAMINATIONS		PERRECTAL EXAMINATION				
	RS EXAMINATIONS		PERVEGINAL EXAMINATION				
INVESTIGATIONS	LAB. INVESTIGATIONS			X-RAY			
		HB		CHEST			
		BT		ABDOMEN			
		CT			ERECT		
		BLOOD COUNT ((WBC)			SUPINE		
		URINE	ALBUMIN	OTHERS			
			SUGAR	USG			
		BLOOD SUGAR		CT			
		BLOOD UREA		DIAGONIS & PLAN AFTER INVESTIGATION			
		SERUM CREATININE					
	SERUM ELECTROLYTES						

TREATMENT DETAILS	OPERATIVE FINDINGS			PROCEDURE DONE		
	SITE OF BOWEL INVOLVEMENT			ANASTAMOSIS		
		SMAL BOWEL			END TO END	
		LARGE BOWEL			END TO SIDE	
	LENGTH OF BOWEL AFFECTED				SIDE SIDE	
		LESS THAN 100 cm		STOMA		
		MORE THAN 100 cm			ENTEROTOMY	
	PRESENCE OF PEFORATION				END	
		YES			LOOP	
		NO			COLOSTOMY	
	PRESENCE OF FECAL PERITONITIS				END	
		YES			LOOP	
		NO		OTHERS		
	UNDERLYING PATHOLOGY					
		BAND				
		ADHESIONS				
		HERNIA				
		VOLVULUS				
		MVD				
	MVD			INTRA OPERATIVE COMPLICATIONS		
	MALIGNANCY				ANASTHESIA	
OTHERS				BLEEDING		
POST OPERATIVE EVENTS	POST OPERATIVE COMPLICATION			FINAL OUTCOME		
		DELAYED POST OP., RECOVERY			COMPLETE RECOVERY	
		WOUND INFECTION			DEATH	
		INTRA ABDOMINAL ABCESS		FOLLOW UP		
		BURST ABDOMEN				
		ANASTAMOTIC LEAK				
		RESPRIATORY INFECTION				
		RENAL INSUFFICIENCY		HPE		
		ELECTROLITE IMBALANCE				
		METABOLIC DERANGMENT				
		SEPTICEMIA				
		OTHERS				

ABBREVIATION

MVD	Mesenteric Vascular Disease
SLE	Systemic Lupus Erythematosus
TAO	Thrombo Angitis Obliterans
DM	Diabetes Mellitus
HT	Hypertension
PT	Pulmonary Tuberculosis
NSAID	Non-Steroidal Anti-inflammatory Drug
PID	Pelvic Inflammatory Disease
ARDS	Acute Respiratory Distress Syndrome
COPD	Chronic Obstructive Pulmonary Disease
PRF	Pre-Renal Failure
SIH	Strangulated Inguinal Hernia
SFH	Strangulated Femoral Hernia
SB	Small Bowel

LB	Large Bowel
Hb	Hemoglobin
PCV	Packed Cell Volume
WBC	White Blood Count
USG	Ultrasonogram
CT	Computerised Tomography

Turnitin Document Viewer - Google Chrome
https://www.turnitin.com/dv?s=1&o=293543124&u=1014644602&istudent_user=1&lang=en_us&

TNMGRMU APRIL 2013 EXAMINA... Medical - DUE 31-Dec-2012 What's New

Originality GradeMark PeerMark

Analysis of 50 cases of gangrenous bowel disease
BY UMA MAHESWARI 22101183 M.S. GENERAL SURGERY

turnitin 6% SIMILAR OUT OF 0

CERTIFICATE

This is to certify that dissertation entitled ANALYSIS OF 50 CASES OF GANGRENOUS BOWEL DISEASES' is a bonafide record of work done by Dr. T. UMA MAHESWARI, in the Department of General Surgery, Thanjavur Medical College, Thanjavur, during her Post Graduate Course from 2010-13. This is submitted in partial fulfillment for the award of M.S., DEGREE EXAMINATION - BRANCH I (GENERAL SURGERY) to be held in March 2012 under The Tamilnadu Dr. M.G.R. Medical University, Chennai.

Dr. M. ELANGO VAN, M.S.,
Unit Chief
Department of surgery,
Thanjavur Medical College,
Thanjavur.

Dr. V. BALAKRISHNAN, M.S.,
Professor and Head,
Department of surgery,
Thanjavur Medical College,
Thanjavur.

DEAN,
Thanjavur Medical College,
Thanjavur.

Match Overview

1	www.cgb.ki.se Internet source	<1%
2	www.cls.usouthal.edu Internet source	<1%
3	Kaleya, Ronald, and Sc... Publication	<1%
4	www.hkcm.com Internet source	<1%
5	Boley, S.J.. "Ischemic... Publication	<1%
6	www.wjgnet.com Internet source	<1%
7	N. C. Gourtsoyiannis. ... Publication	<1%
8	uconndocs.uchc.edu Internet source	<1%
9	beta.lumhs.edu.pk Internet source	<1%

PAGE: 1 OF 140

22:12
17-12-2012